PB86-177581

# Acid Deposition, Trace Contaminants and Their Indirect Human Health Effects Research Needs

New York State Dept. of Environmental
Conservation, Albany

Prepared for

Corvallis Environmental Research Lab., OR

Mar 86

U.S. Department of Commerce
National Technical Information Service
NTIS

EPA/600/9-86/002
March 1986

PB86-177581

WORKSHOP PROCEEDINGS

# ACIDIC DEPOSITION, TRACE CONTAMINANTS AND THEIR INDIRECT HUMAN HEALTH EFFECTS: RESEARCH NEEDS

held

June 19-22, 1984

at

Topridge Conference Center
Paul Smith's, New York

Editor

Scott O. Quinn
Bureau of Water Research
New York State Department of Environmental Conservation

Assistant Editor

Naomi Bloomfield, M.D.
Albany Medical College

Sponsors

U.S. Environmental Protection Agency
New York State Department of Environmental Conservation
Albany Medical College

Project Officer

Robert T. Lackey
U.S. Environmental Protection Agency
Environmental Research Laboratory - Corvallis
Corvallis, Oregon

ENVIRONMENTAL RESEARCH LABORATORY - CORVALLIS
CORVALLIS, OREGON

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U.S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

## TECHNICAL REPORT DATA

*(Please read Instructions on the reverse before completing)*

**1. REPORT NO.**
EPA/600/9-86/002

**2.**

**3. RECIPIENT'S ACCESSION NO.**
PB86 177581 /AS

**4. TITLE AND SUBTITLE**
WORKSHOP PROCEEDINGS: Acid Deposition, Trace Contaminants & Their Indirect Human Health Effects: Research Needs

**5. REPORT DATE**
March 1986

**6. PERFORMING ORGANIZATION CODE**

**7. AUTHOR(S)**
S.O.Quinn & N. Bloomfield, eds.

**8. PERFORMING ORGANIZATION REPORT NO.**

**9. PERFORMING ORGANIZATION NAME AND ADDRESS**
Bureau of Water Research, NY Dept of Environmental
Conservation, 50 Wolf Rd, Albany, NY 12233-0001

**10. PROGRAM ELEMENT NO.**

**11. CONTRACT/GRANT NO.**
P04B1201NAEX

**12. SPONSORING AGENCY NAME AND ADDRESS**
Environmental Research Laboratory
Office of Research and Development
U.S.Environmental Protection Agency
Corvallis, OR 97333

**13. TYPE OF REPORT AND PERIOD COVERED**
Proceedings (workshop)

**14. SPONSORING AGENCY CODE**
EPA 600/02

**15. SUPPLEMENTARY NOTES**
Project Officer: Robert Lackey, FTS 420-4806

**16. ABSTRACT**

A workshop to examine the research needs for acidic deposition, trace contaminants and their indirect human health effects was held in the Adirondack Mountains of NY, June 19-22, 1984. The meeting was co-sponsored by the USA EPA, the NY State Dept of Environmental Conservation, and the Albany Medical College.

The purpose of the workshop was to: 1) review the current knowledge base; and 2) propose a research agenda concerning the relationship between acidic deposition and altered biogeochemical cycling of trace contaminants, as related specifically to increased human exposure(post-deposition).

The conference was organized into 4 groups, each focusing on one aspect of the acidic deposition phenomenon. Working groups in atmospheric processes, biogeochemistry, bioaccumulation and human health effects developed state-of-the-art summaries concerning acidic deposition effects, summarized known and unknown aspects of the acidic deposition phenomenon and identified research needs.

The groups selected specific trace contaminants for study. They concluded that acidic deposition may influence human exposure to trace contaminants via two main pathways: 1) the accumulation of contaminants in the food chain leading to man; and 2) the contamination of drinking water. Participants determined that contaminants requiring special attention are mercury, lead, aluminum, and cadmium. This decision was based on several factors, including human toxicity and potential for human exposure.

**17. KEY WORDS AND DOCUMENT ANALYSIS**

| a. DESCRIPTORS | b. IDENTIFIERS/OPEN ENDED TERMS | c. COSATI Field/Group |
|---|---|---|
| | | |

| | | |
|---|---|---|
| **18. DISTRIBUTION STATEMENT**<br>Release to Public | **19. SECURITY CLASS** *(This Report)*<br>Unclassified | **21. NO. OF PAGES**<br>89 |
| | **20. SECURITY CLASS** *(This page)*<br>Unclassified | **22. PRICE** |

## DISCLAIMER

This workshop proceedings has been funded as part of the National Acid Precipitation Assessment Program in part by the U.S. Environmental Protection Agency under contract P.O. #4B1201NAEX to the N.Y.S. Department of Environmental Conservation. This proceedings has been subject to the EPA's peer and administrative review, and it has been approved for publication as an EPA document. Although it has been approved for publication as an EPA document, because it is a workshop proceedings, it does not necessarily reflect the view of the Agency and no official endorsement should be inferred.

# PREFACE

A workshop to examine the research needs for acidic deposition, trace contaminants and their indirect human health effects was held at the Topridge Conference Center in the Adirondack Mountains of New York State during June 19-22, 1984. The meeting was co-sponsored by the United States Environmental Protection Agency, the New York State Department of Environmental Conservation, and the Albany Medical College.

The purpose of the workshop was to: (1) review the current knowledge base; and (2) propose a research agenda concerning the relationship between acidic deposition and altered biogeochemical cycling of trace contaminants, as related specifically to increased human exposure (post-deposition).

Workshop attendees were selected to represent a broad spectrum of scientists with an established record of expertise in acidic deposition research. Most participants have been actively involved in national and international efforts to develop research programs with respect to acidic deposition.

The conference was organized into four working groups, each focusing on one aspect of the acidic deposition phenomenon. Working groups in atmospheric processes, biogeochemistry, bioaccumulation and human health effects developed state-of-the-art summaries concerning acidic deposition effects. Each working group summarized known and unknown aspects of the acidic deposition phenomenon and identified research needs.

The four working groups collaborated to select specific trace contaminants for study. They concluded that acidic deposition may influence human exposure to trace contaminants via two main pathways: (1) the accumulation of contaminants in the food chain leading to man; and (2) the contamination of drinking water. After much discussion it was decided that those contaminants requiring special attention are mercury, lead, aluminum, and cadmium. This decision was based on several factors, including human toxicity and potential for human exposure.

The research agenda which follows addresses gaps in the data concerning the impacts of acidic deposition on the biogeochemical cycling of important trace contaminants. The research needs identified, therefore, are those required to elucidate the human health effects indirectly exacerbated by acidic deposition.

## TABLE OF CONTENTS

Page
PREFACE.......................................................... iii
WORKSHOP PARTICIPANTS............................................ ix

Section

I. CURRENT STATE OF KNOWLEDGE

1. Atmospheric Processes........................................ 1
 1.1 General Introduction....................................... 1
  1.1.1 Emissions............................................... 1
  1.1.2 Transport............................................... 3
  1.1.3 Transformation.......................................... 3
  1.1.4 Deposition.............................................. 3
  1.1.5 Measurements............................................ 5
 1.2 Mercury.................................................... 5
  1.2.1 Emissions............................................... 5
  1.2.2 Transport............................................... 5
  1.2.3 Deposition.............................................. 5
 1.3 Lead....................................................... 10
  1.3.1 Emissions............................................... 10
  1.3.2 Transport............................................... 10
  1.3.3 Deposition.............................................. 10
 1.5 Aluminum................................................... 10
  1.5.1 Emissions............................................... 10
  1.5.2 Transport............................................... 10
  1.5.3 Deposition.............................................. 10
 1.4 Cadmium.................................................... 10
  1.4.1 Emissions............................................... 10
  1.4.2 Transport............................................... 10
  1.4.3 Deposition.............................................. 11

2. Biogeochemistry.......................................... 11

2.1 General Introduction........................... 11

2.2 Mercury.................................................. 13

2.2.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering.................................. 13

2.2.2 Mercury Mobilization from Watershed and Sediments.............................. 13

2.2.3 Influence of Acidification on Mercury Speciation................................. 13

2.2.4 Effect of Acidification on Microbial Transformations of Mercury.................. 14

2.2.5 Effects of Acid Water and Increased Atmospheric Loading on Mercury Levels in Water Distribution Systems.............. 15

2.3 Lead................................................... 15

2.3.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering.................................. 15

2.3.2 Lead Mobilization from Watershed and Sediments.............................. 15

2.3.3 Influence of Acidification on Lead Speciation................................. 16

2.3.4 Effect of Acidification on Microbial Transformations of Lead.................... 16

2.3.5 Effect of Acid Water and Increased Atmospheric Loading on Lead Levels in Water Distribution Systems.................. 16

2.4 Aluminum............................................ 16

2.4.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering.................................. 16

2.4.2 Aluminum Mobilization from Watershed and Sediments.............................. 16

2.4.3 Influence of Acidification on Aluminum Speciation.................................. 17

2.4.4 Effect of Acidification on Microbial Transformations of Aluminum................ 17

2.4.5 Effect of Acid Water and Increased Atmospheric Loading on Aluminum Levels in Water Distribution Systems.............. 17

2.5 Cadmium............................................... 17

2.5.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering................................ 17

2.5.2 Cadmium Mobilization from Watershed and Sediments............................ 17

2.5.3 Influence of Acidification on Cadmium Speciation................................ 18

2.5.4 Effect of Acidification on Microbial Transformations of Cadmium................. 18

2.5.5 Effect of Acid Water and Increased Atmospheric Loading on Cadmium Levels in Water Distribution Systems.............. 18

3. Bioaccumulation........................................ 18

3.1 General Introduction................................ 18

3.2 Mercury............................................. 19

3.2.1 Accumulation of Mercury by Fish............ 19

3.2.2 Effects of Acidification on Mercury Bioaccumulation by Fish................... 19

3.2.3 Effects of Productivity on Mercury Uptake by Fish............................. 23

3.2.4 Effects of Chemical Neutralization of Lakes on Mercury Contamination of Fish..... 24

3.3 Other Metals......................................... 24

4. Indirect Health Effects................................. 24

4.1 General Introduction................................ 24

4.2 Mercury............................................. 27

4.2.1 Environmental Exposure and Uptake by Humans.................................. 27
- 4.2.1.1 Concentrations in Water.......... 27
- 4.2.1.2 Concentrations in Aquatic Biota.. 28
- 4.2.1.3 Human Uptake and Risk Assessment....................... 28

4.2.2 Effects on Human Health.................... 31
- 4.2.2.1 Metabolism in Humans............. 31
- 4.2.2.2 Effects and Dose-Response in Humans........................ 33

4.3 Lead.............................................. 35

4.3.1 Environmental Exposure and Uptake by Humans................................. 35
- 4.3.1.1 Concentrations in Water.......... 35
- 4.3.1.2 Concentrations in Aquatic Biota.. 36
- 4.3.1.3 Human Uptake and Risk Assessment....................... 36

4.3.2 Effects on Human Health.................... 40
- 4.3.2.1 Metabolism in Humans............. 40
- 4.3.2.2 Effects and Dose-Response in Humans........................ 42

4.4 Aluminum.......................................... 47

4.4.1 Environmental Exposure and Uptake by Humans................................... 47
- 4.4.1.1 Concentrations in Water.......... 47
- 4.4.1.2 Concentrations in Aquatic Biota.. 48
- 4.4.1.3 Human Uptake and Risk Assessment....................... 48

4.4.2 Effects on Human Health.................... 49
- 4.4.2.1 Metabolism in Humans............. 49
- 4.4.2.2 Effects and Dose-Response in Humans.......................... 49

4.5 Cadmium........................................... 50

4.5.1 Environmental Exposure and Uptake by Humans.................................... 50

4.5.1.1 Concentrations in Water.......... 50

4.5.1.2 Concentrations in Aquatic Biota.. 50

4.5.1.3 Human Uptake and Risk Assessment......................... 50

4.5.2 Effects on Human Health.................... 51

4.5.2.1 Metabolism in Humans............. 51

4.5.2.2 Effects and Dose-Response in Humans.......................... 51

4.6 Other Trace Contaminants.......................... 51

4.6.1 Asbestos................................... 51

4.6.2 Nitrates................................... 52

II. SUMMARY OF RESEARCH RECOMMENDATIONS

Atmospheric Processes........................................... 53

Biogeochemistry................................................. 53

Bioaccumulation................................................. 55

Indirect Health Effects.......................................... 55

III. LITERATURE CITED

References...................................................... 58

Appendices

A. AGENDA FOR WORKSHOP............................................. 74

B. ACKNOWLEDGEMENTS................................................ 76

# WORKSHOP PARTICIPANTS

## ATMOSPHERIC PROCESSES

### Chairman

Perry J. Samson, Ph.D.
University of Michigan
Department of Atmospheric and
Ocean Science
2213 Space Research Building
Ann Arbor, Michigan 48109

Kenneth T. Knapp, Ph.D.
Supervisory Research Chemist
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

Cliff I. Davidson, Ph.D.
Assistant Professor of Civil
Engineering & Public Policy
Carnegie-Mellon University
Pittsburgh, Pennsylvania 15213

Liquat Husain, Ph.D.
Director, Chemical Sciences Lab.
Center for Labs and Research
N.Y.S Department of Health
Room D-539
Albany, New York 12201

Thomas M. Church, Ph.D.
College of Marine Sciences
University of Delaware
Newark, Delaware 19716

Philip J. Galvin, Ph.D.
Division of Air Resources
N.Y.S. Department of
Environmental Conservation
50 Wolf Road
Albany, New York 12233-0001

## BIOGEOCHEMISTRY

### Chairman

Peter G.C. Campbell, Ph.D.
Universite of Quebec
Institut national de la
recherche scientifique
C.P. 7500
Ste.-Foy, Quebec, Canada G1V 4C7

Stephan A. Norton, Ph.D.
Department of Geological Sciences
University of Maine at Orono
110 Boardman Hall
Orono, Maine 04469

Hans Hultberg, Ph.D.
Research Group Leader
Swedish Environmental Research
Institute
P.O. Box 5207
S-402 24 Gothenburg 5, Sweden

Michael R. Winfrey, Ph.D.
Department of Biology
University of Wisconsin
La Crosse, Wisconsin 54601

## BIOACCUMULATION

### Chairman

James G. Wiener, Ph.D.
U.S. Fish & Wildlife Service
Columbia Nat. Fish. Res. Lab.
Field Research Station
P.O. Box 936
La Crosse, Wisconsin 54601

Pamela M. Stokes, Ph.D.
Institute for Environ. Studies
University of Toronto
Haultrain Building
Toronto, Ontario, Canada M5S 1A6

## BIOACCUMULATION (con't.)

Ronald J. Sloan, Ph.D.
Division of Fish & Wildlife
N.Y.S. Dept. Environmental
Conservation
50 Wolf Road
Albany, New York 12233-0001

## HUMAN HEALTH EFFECTS

### Chairman

Thomas W. Clarkson, Ph.D.
Division of Toxicology
University of Rochester
School of Medicine
P.O. Box RBB
Rochester, New York 14642

Magnus Piscator, M.D.
Department of Environmental Hygiene
Karolinska Institute
P.O. Box 60400
S-104 01 Stockholm, Sweden

David A. Bennett, Ph.D.
Director
Acid Deposition Assessment Staff
U.S. Env. Protection Agency
401 M Street, SW (RD-676)
Washington, D.C. 20460

Harvey Olem, Ph.D.
Tennessee Valley Authority
248 401 Building
Chattanooga, Tennessee 37401

Shadad Joshi, M.D.
Albany Medical Center
New Scotland Avenue
Albany, New York 12209

Gunar Nordberg, M.D.
Professor
Dept. of Environmental Medicine
Umea University
S-901 87 Umea, Sweden

Robert G. Miller, Ph.D.
Health Effects Research Lab.
U.S. Env. Protection Agency
26 West St. Claire Street
Cincinnati, Ohio 45268

Robert A. Goyer, M.D.
National Institute of
Environmental Health Sciences
P.O. Box 12233
Research Triangle Park, NC 27709

Paul D. Moskowitz, Ph.D.
Health Effects Assessment
Brookhaven National Laboratory
Upton, New York 11973

Michael McDonald, Ph.D.
Center for Limnology
680 N. Park Street
University of Wisconsin
Madison, Wisconsin 53706

## WORKSHOP ADMINISTRATION

Scott O. Quinn
Bureau of Water Research
N.Y.S. Department of
Environmental Conservation
Room 317
50 Wolf Road
Albany, New York 12233-0001

Naomi Bloomfield, M.D.
Albany Medical College
New Scotland Avenue
Albany, New York 12209

## WORKSHOP ADMINISTRATION (con't.)

Joseph M. Eilers
U.S. Environmental Protection Agency
200 SW 35th Street
Corvallis, Oregon 97333

# SECTION I: CURRENT STATE OF KNOWLEDGE

## 1. ATMOSPHERIC PROCESSES (P.J. Samson, T.M. Church, C.I. Davidson, P.J. Galvin, L. Husain, K.T. Knapp)

### 1.1 GENERAL INTRODUCTION

The atmosphere plays a major role in the movement of trace elements in the environment. Trace elements are known to be transported in the atmosphere from their source to their eventual deposition. This direct transfer of material to the soil may be an important input of some elements including sulfur, selenium, lead and cadmium which have large anthropogenic emissions. For other trace elements such as aluminum, the importance of direct atmospheric input is thought to be secondary to the in-soil mobilization of the element by atmospheric acids (see Section 2.). This section briefly discusses the role of the atmosphere in transporting and depositing of both trace elements and atmospheric acids, and prioritizes the research agenda needed to gain a firmer understanding of the atmospheric input of trace elements into the environment. Emphasis is given to the elements mercury, lead, aluminum,and cadmium because these have been identified as potentially toxic metals in the environment. Sulfur is also discussed inasmuch as the acidity associated with sulfur oxides is believed to be responsible for mobilizing trace metals in the soil.

The atmospheric input of both trace elements and acidic materials into the environment is the result of four major processes: emission, transport, transformation and deposition. Each of the processes can be investigated individually to identify areas where additional knowledge is required.

1.1.1 Emissions -- Data about the emissions of trace elements into the atmosphere are limited in many aspects. Almost all studies of trace element emissions have not addressed the form, size distribution, or speciation of the emitted material. Only emissions of sulfur dioxide, the precursor for particulate sulfate, have been quantified with any confidence.

Lantzy and Mackenzie (1979) and Galloway et al. (1980) have estimated the global magnitude of natural and anthropogenic emissions of many trace elements. Table 1-1 lists their estimates of the global emission rates for mercury, lead and cadmium. On this scale it is obvious that the atmospheric source of these elements is largely anthropogenic, though natural emissions of mercury in the vapor phase are relatively large. The more recent estimates for global emission of mercury by Lindquist et al. (1984) appears to be about an order of magnitude lower than those estimated by Lantzy and MacKenzie. Fitzgerald et al. (1983) have estimated global emissions at $8 \times 10^6$ kg $y^{-1}$ using new data on biological emissions from the oceans. Thus the estimation of global mercury emissions is still debated.

Data for specific parts of the globe and for specific industries is relatively unknown. Pacyna et al. (1984) have estimated the emissions rate of several trace elements from European sources by source type. Table 1-2 lists their estimates for lead and cadmium for 1979. Data for sized particles from coal-fired and oil-fired power plants, pulp and paper plants and sludge incineration have been reported (Bennett and Knapp, 1978; Henry, 1979; Henry and Knapp, 1980) but these data did not include several of the important trace elements. Few data exist on the elemental content of non-point source emissions

Table 1-1. Estimates of natural and anthropogenic global annual emission rates for mercury, lead and cadmium in units of $10^6$ kg $y^{-1}$ as derived from Galloway et al., (1980). Numbers in parentheses represent estimates of vapor emissions.

| | Natural | Anthropogenic |
|---|---|---|
| Mercury | 0.4 (25) | 11 |
| Lead | 5.9 | 2,000 |
| Cadmium | 0.3 | 6 |

Table 1-2. European emissions of trace elements in 1979 as estimated by Pacyna et al., (1984) in units of $10^6$ kg $y^{-1}$.

| | Cd | Pb |
|---|---|---|
| Coal Combustion | 0.15 | 1.67 |
| Oil Combustion | 0.11 | 1.12 |
| Wood Combustion | 0.025 | 0.56 |
| Gasoline Combustion | 0.031 | 74.3 |
| Mining | 0.001 | 1.09 |
| Copper-Nickel Smelters | 0.60 | 9.25 |
| Zinc-Cadmium Smelters | 1.55 | 7.88 |
| Lead Smelters | 0.008 | 10.45 |
| Manufacturing | 0.058 | 14.66 |
| Refuse Incinerators | 0.84 | 0.80 |
| Phosphate Fertilizers | 0.027 | 0.01 |
| Cement Production | 0.015 | 0.75 |

which could be a major contribution of certain trace elements in the environment.

1.1.2 Transport -- Atmospheric transport of pollutants from emission to eventual deposition is dependent upon the speed of the transporting winds and the rate of removal from the atmosphere. The removal of pollutants occurs by dry or wet deposition. The pollutant can be deposited at the surface through sedimentation or impaction and uptake (dry deposition) or can become incorporated into hydrometeors (cloud droplets, raindrops, or snowflakes) and be deposited to the surface (wet deposition). In general, the distance the pollutant travels before it deposits depends, in part, on the height of injection of the material into the atmosphere. This is because, in general, the wind speed increases with height and hence the pollutant is carried further, and also because the elevated releases require a longer time for the plume to be mixed to the surface and dry deposited.

It is useful to compare the transport of trace elements to that of the more-studied sulfur oxides to identify the areas where our understanding of trace element transport is still poor. This section also serves to illustrate the transport scales of sulfur oxides responsible for the production of acidic precipitation.

Sulfur oxides are emitted largely as gaseous sulfur dioxide ($SO_2$) though it is estimated that 1 to 5% of the sulfur oxide is emitted directly as sulfate. The percentage of this primary sulfate emission is thought to be highest for oil-fueled sources. While the bulk of the sulfur is transported away from the source area, a fraction of the sulfur will be oxidized into sulfate ($SO_4$) and a fraction will be removed as $SO_2$ through dry deposition to within 50 km of the source.

The distance the pollutant travels before depositing is dictated in part by the physical and chemical characteristics of the particle being transported. Particulate sulfate, for example, is created through a gas-to-particle conversion process which yields very small aerosol particles. These particles will collide and grow to a size of between 0.1 to 1.0 μm. In this size range, the particles have very small deposition velocities and can travel very long distances without significant losses due to dry deposition.

1.1.3 Transformation -- Transformation involves a chemical change in the emitted species. Such changes can affect the efficiency with which the material is deposited by precipitation and the residence time of the material. Chemical transformation is known to be important in the formation of atmospheric acids.

Most trace elements enter the atmosphere directly in a particulate form or very rapidly condense into particles as the hot gaseous effluent cools. An exception to this occurs with mercury and possibly cadmium (Galloway et al., 1980). The reactivity of these trace elements is thought to be small compared to sulfur oxides (Lindquist et al., 1984).

1.1.4 Deposition — Trace elements are deposited at the earth's surface by "wet" or "dry" processes. Wet deposition is usually confined to precipitation (rain or snow) although dew and fog condensation are sometimes included. Dry deposition includes processes such as sedimentation, interception, inertial impaction, and diffusion; thermophoratic and electrostatic forces may also be important in some instances. In all but very dry areas, wet deposition accounts for at least half of the total deposition for those trace elements predominantly composed of submicron particles. For trace elements largely dominated by natural crustal sources (e.g. Fe, Mn, and possibly Cu) dry deposition is as great or even greater than wet deposition on an annual basis as evidenced from secular increases in trace elements to aquatic systems during differential transport by wind conditions. For other trace elements which have predominant inventories in the gaseous phase (e.g. Hg, some metalloids), dry deposition via gas exchange with aquatic surfaces appears important. However, most estimates of total deposition of trace elements rely on our very limited knowledge of deposition velocities, and the ambient atmosphere concentrations of those elements. Total deposition estimates based on soil profiles are also fraught with geochemical uncertainties of diagenetic post-depositional mobilities of many trace elements (see Section 2.1).

Measurement of trace elements in precipitation at even remote ambient concentrations is within the current state of analytical technique (eg. Church et al., 1985; Davidson et al., 1984a). However, extrapolation of such trace element concentrations to wet deposition assumes minor contamination during collection and the use of adequate procedures to quantify the lack of background concentration in blank samples. Procedures for collection of trace elements in precipitation have been shown to require strict protocols of trace element-free and clean collector equipment, containers, and reagents. These protocols need to be established for individual areas and needs, but are now within the state of the art for collection, processing, and analysis of trace elements in natural waters at ultra trace concentrations. Nevertheless, extrapolation of trace element concentrations in precipitation to actual wet deposition still has not clearly resolved particulate versus dissolved trace element burdens or speciation.

Measurement of trace element dry deposition is far more difficult than measurement of wet deposition. Examples of dry flux assessment techniques include exposure of surrogate surfaces, vegetation washing, and micrometeorological methods. Exposure of surrogate surfaces is convenient and inexpensive, but the technique has been criticized due to the inability to simulate natural surfaces (Hicks et al., 1980). Recent data has suggested the possible utility of the method for assessing fluxes of large particles (Davidson et al., 1984b).

Vegetation washing may provide direct deposition data on the surfaces of interest. However, large variability among samples, and interferences caused by leaching of internal material, limit the utility of the method for routine monitoring (Lindberg et al., 1982).

Finally, examples of micrometeorological techniques include measurement of concentration gradients (Garland, 1978), eddy correlation (Wesley et al., 1977), and variance techniques (Wesley and Hicks, 1978). These methods may provide estimates of vapor and submicron particle fluxes, but the strict fetch and analytical precision requirements, as well as the expense of operating such systems, limit their utility (Hicks et al., 1980). It is apparent that the limitations of currently available techniques prevent the acquisition of reliable trace element dry deposition data on a routine basis.

1.1.5 Measurements -- Measured atmospheric concentrations of trace metals have been summarized by Galloway et al. (1980). A similar compilation of concentrations in precipitation has been presented by Galloway et al. (1982). They report a range of concentrations in the atmosphere and precipitation as illustrated in Figures 1-1 and 1-2, respectively, for mercury; Figures 1-3 and 1-4 for lead and Figures 1-5 and 1-6 for cadmium. As expected, the highest mean concentrations measured are for urban areas. The "remote" locations cited have concentrations from one to several orders of magnitude lower than "urban" samples.

Lindberg (1982) has investigated the variations in lead concentrations in rain. He shows that lead is higher in the summer months in response to increased air stagnation and lower precipitation volume (hence less dilution) during that season. Husain and Samson (1979) illustrated that in a remote location during the summer the concentrations of some elements are governed by the wind flow. At Whiteface Mountain, New York extensive measurements have shown that lead and several other elements concentrations are enhanced approximately four-fold when windflow is from the Midwestern United States (Husain, 1984). Atmospheric transport of trace metals from mid-latitude source regions to the Arctic has been documented by several investigators (Rahn, 1981; Barrie et al., 1981; Davidson et al., 1984a). Thus it is obvious that those trace metals which are found as submicron particles are being transported long distances from their source.

The acidity of precipitation may also be contributing to the mobilization of metals in the soil (see Section 2.1). Current measurements of hydrogen ion concentration generally show a maximum deposition rate in the upper Ohio River basin. Figure 1-7 shows this distribution for the United States and Canada in units of pH.

## 1.2 MERCURY

1.2.1 Emissions -- Much of the emissions data for trace elements is derived from analyses of collected particles (fly ash). Thus, little data exist on the emissions of mercury into the atmosphere because a sizable fraction of the emitted material is in the vapor phase (Lindquist et al., 1984). Likewise, although anthropogenic emissions of mercury are significant, there are uncertainties about the natural emission rates.

1.2.2 Transport -- The atmospheric transport of mercury has also not been adequately defined because of the lack of vapor-phase data. Husain and Samson (1979) found that particulate mercury behaved like no other trace element, having its highest concentrations at times most other elements were significantly below average.

1.2.3 Deposition -- Mercury in the environment is partially due to direct atmospheric input. This fraction is hard to quantify because of the lack of reliable data. The increased groundwater input of mercury to aquatic systems is augmented by increased acidification of historically deposited mercury. This mercury has accumulated in the soil profiles over recent industrial times and may be released into the aquatic systems as the pH of the precipitation decreases (see Sections 2.2.1 and 2.2.2).

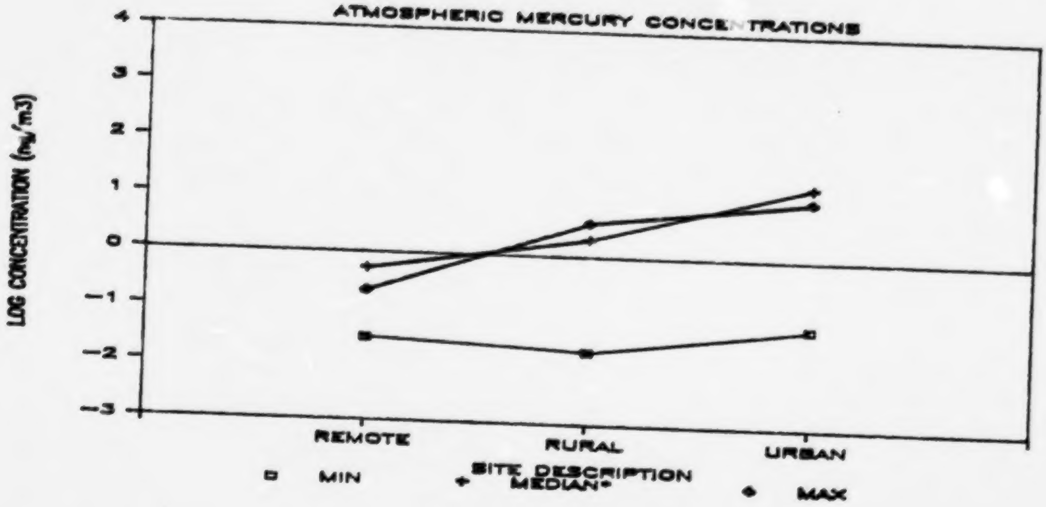

Figure 1-1. Minimum, median and maximum concentrations of airborne mercury found in the literature by Galloway et al. (1980) for remote, rural and urban locations. Median concentrations are for total Hg while max and min include particulate Hg only.

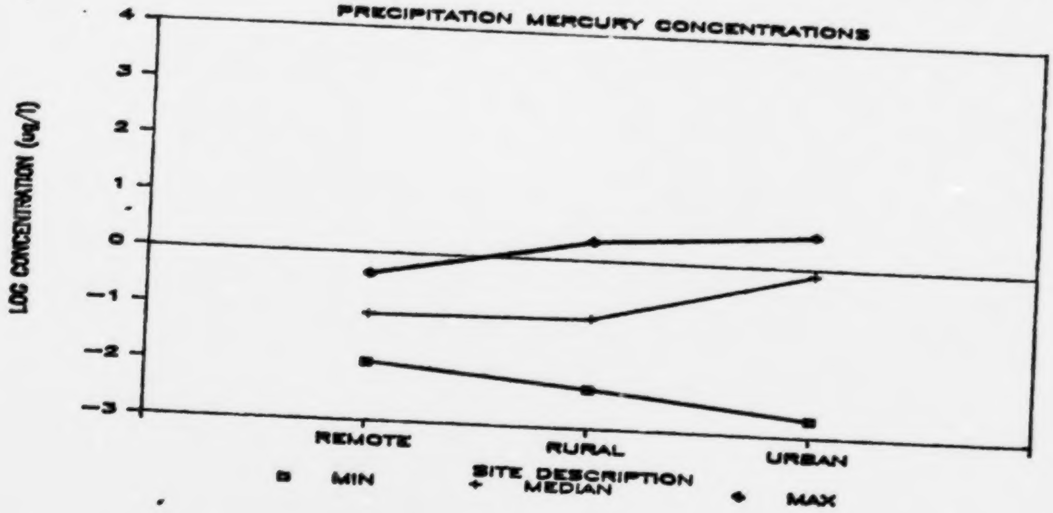

Figure 1-2. Minimum, median and maximum concentrations of mercury in precipitation found in the literature by Galloway et al. (1982) for remote, rural and urban locations.

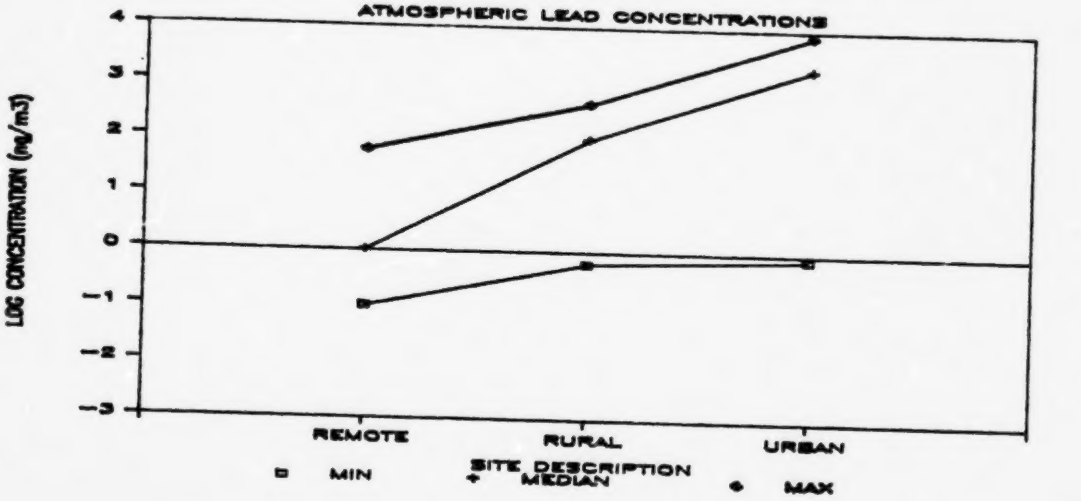

Figure 1-3. Minimum, median and maximum concentrations of airborne lead found in the literature by Galloway et al. (1980) for remote, rural and urban locations.

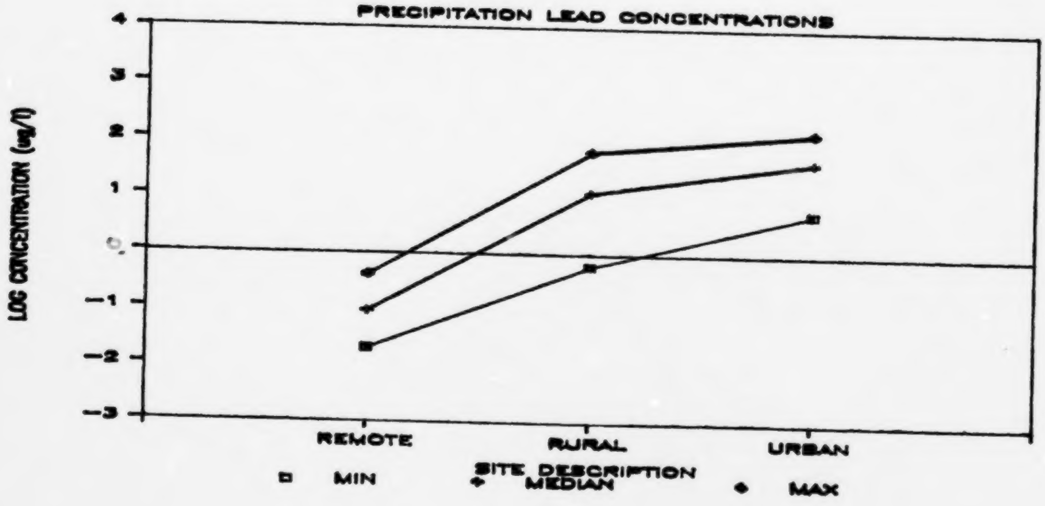

Figure 1-4. Minimum, median and maximum concentrations of lead in precipitation found in the literature by Galloway et al. (1982) for remote, rural and urban locations.

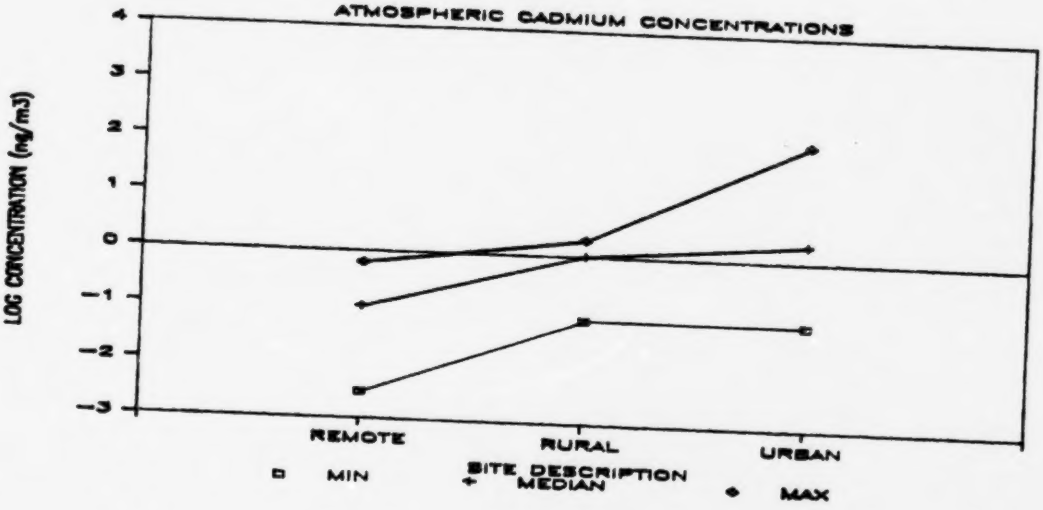

Figure 1-5. Minimum, median and maximum concentrations of airborne cadmium found in the literature by Galloway et al. (1980) for remote, rural and urban locations.

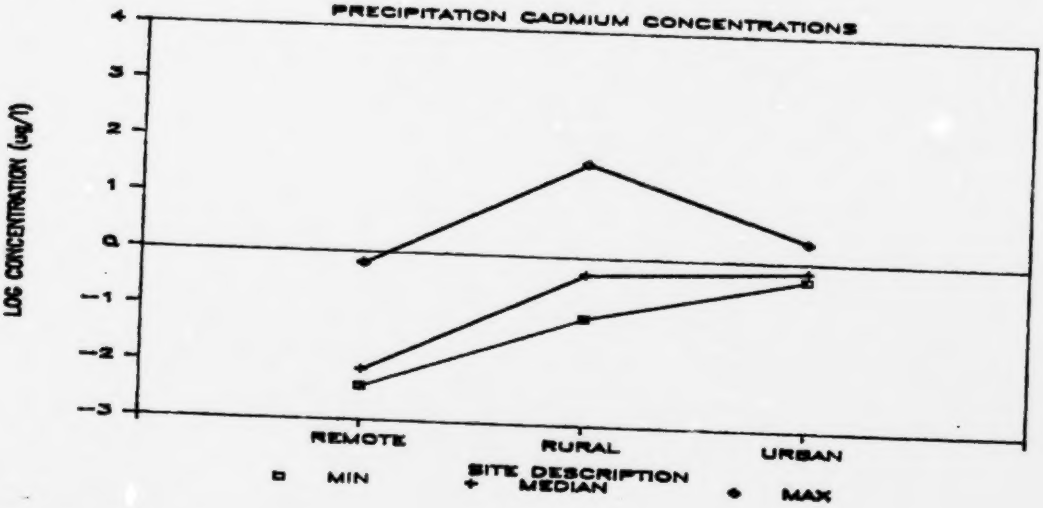

Figure 1-6. Minimum, median and maximum concentrations of cadmium in precipitation found in the literature by Galloway et al. (1982) for remote, rural and urban locations.

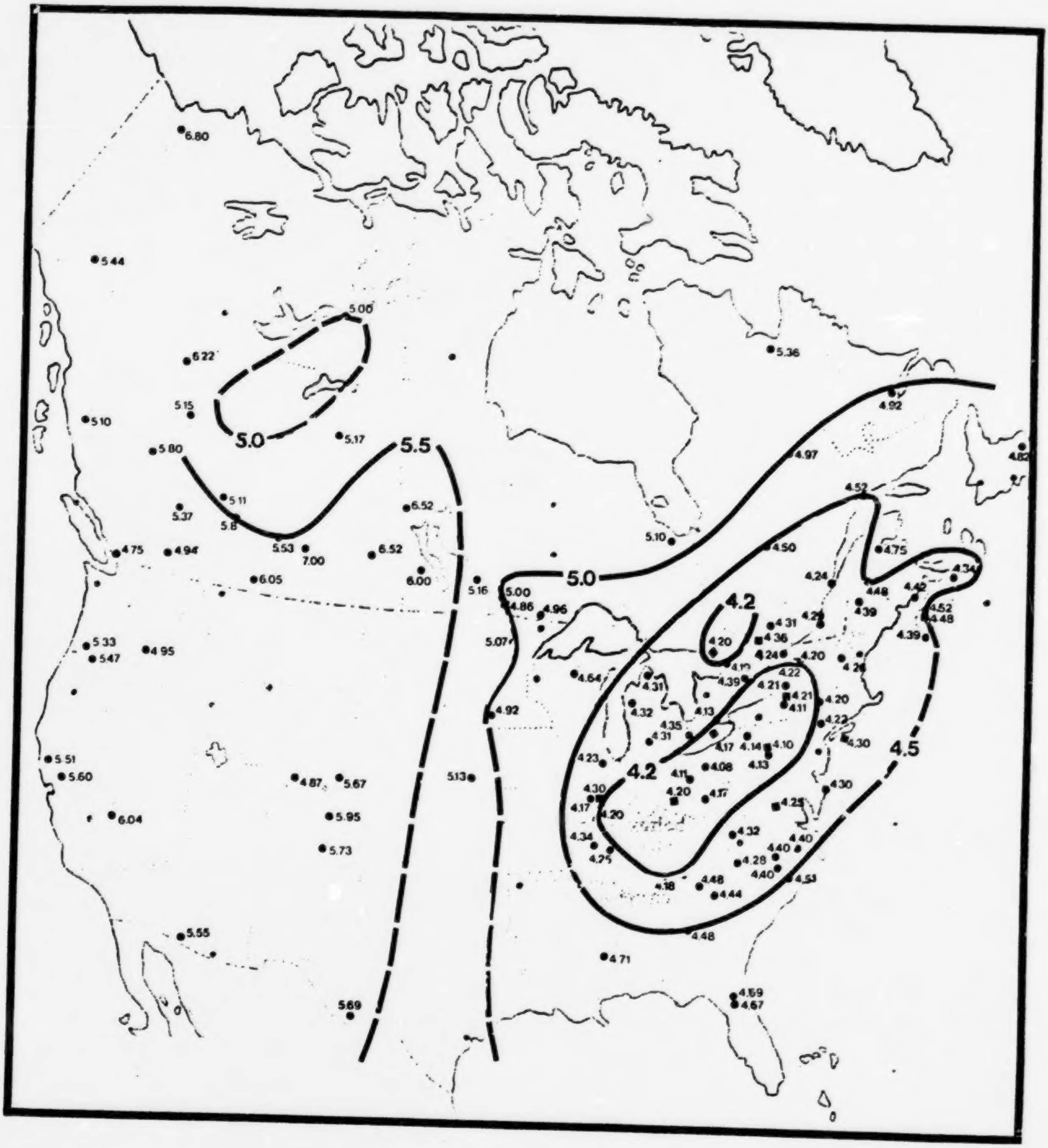
6.80
5.44
5.00
6.22
5.15
5.10
5.80
5.0
5.17
5.5
5.11
5.37
5.8
6.52
4.75
4.94
5.53
7.00
6.52
6.00
6.05
5.16
5.00
4.86
4.96
5.33
5.47
4.95
5.07
4.92
5.51
5.60
4.87
5.67
5.13
6.04
5.95
5.73
5.55
5.69
5.36
4.92
4.97
4.82
4.52
5.10
4.50
4.75
5.0
4.24
4.48
4.34
4.42
4.2
4.39
4.52
4.48
4.31
4.36
4.20
4.24
4.20
4.39
4.64
4.31
4.32
4.13
4.22
4.21
4.21
4.11
4.20
4.22
4.35
4.31
4.17
4.14
4.10
4.13
4.30
4.5
4.23
4.2
4.11
4.08
4.30
4.17
4.20
4.17
4.25
4.30
4.34
4.25
4.32
4.40
4.40
4.28
4.40
4.51
4.18
4.48
4.44
4.48
4.71
4.69
4.67

Figure 1-7. Annual mean value of pH in precipitation weighted by the amount of precipitation in the United States and Canada for 1980 (from Calvert et al., 1983).

## 1.3 LEAD

1.3.1 Emissions -- Compared to mercury the emissions of lead are probably better understood. The emissions of lead are largely the result of vehicular emissions, with smaller amounts from stationary sources such as coal combustion, smelting, and similar industries (Patterson, 1980).

1.3.2 Transport -- The transport of lead in the atmosphere is thought to be similar to other fine particles. The particles generally coagulate to a diameter of 0.1 - 1.0 µm and thus are capable of being transported long distances.

1.3.3 Deposition -- The wet deposition of lead in rural areas of high precipitation is probably dominate over dry deposition. For example, Peirson et al. (1973) have shown that dry deposition of lead accounts for less than 10 percent of the total deposition at a rural site in England. However, dry deposition may play a significant role in areas receiving less precipitation. Roughly equal wet and dry annual fluxes of lead for Walker Branch Watershed, Tennessee have been reported by Lindberg et al. (1982).

## 1.5 ALUMINUM

1.5.1 Emissions -- Aluminum, unlike the other trace metals discussed above, is largely soil derived. There is evidence that aluminum is being mobilized in the soil by the increased acidity of the precipitation.

1.5.2 Transport -- Aluminum is generally composed of larger particles than the other three trace metals discussed in this report. Thus, its ability to be transported long distances is not as great.

1.5.3 Deposition -- Both wet and dry deposition velocities of aluminum are significantly greater than those of lead and cadmium, due to the larger particle sizes associated with aluminum. Davidson et al. (1985b) have reported that dry deposition velocities for aluminum are more than an order of magnitude greater than those of lead or cadmium; Peirson et al. (1973) have reported a factor of three difference.

These latter authors have also reported total annual dry deposition fluxes for aluminum which are approximately one-fifth of the total deposition fluxes at a rural site in England. These values may have been influenced by resuspension of local soil, however, and must be regarded as preliminary.

## 1.4 CADMIUM

1.4.1 Emissions -- Important emission sources of cadmium include smelters and refuse incinerators. Incinerators may become increasingly important as communities deal with their growing need for solid waste disposal. Details of the emissions of cadmium have been discussed by Nriagu (1980).

1.4.2 Transport -- Cadmium in the atmosphere is associated with somewhat larger particles than lead. A recent literature review by Davidson and Osborn (1985a) has shown an average mass median aerodynamic diameter (MMAD) of 1.5 µm, with 25 percent of the mass associated with particles smaller than 0.5 µm and that lead has an average MMAD of 0.53 µm with 51 percent of the mass less than 0.5 µm. Thus, one would expect cadmium to be significantly influenced by long-range transport, although perhaps to a lesser extent than lead.

1.4.3 Deposition -- Less is known about the deposition rates of cadmium then is known about the deposition rates of lead. Lindberg et al. (1982) reported that the annual dry deposition fluxes which are approximately one-fifth of the annual wet deposition in Walker Branch Watershed, Tennessee. Davidson et al. (1985b) report that cadmium dry deposition velocities onto surrogate surfaces are comparable to those of lead in Olympic National Park, Washington. More information is needed before cadmium deposition rates are known with any degree of certainty.

## 2. BIOGEOCHEMISTRY (P.G.C. Campbell, H. Hultberg, S.A. Norton, M.R. Winfrey)

### 2.1 GENERAL INTRODUCTION

Workshop participants considered the relationship between acidic deposition and altered biogeochemical cycling of trace contaminants (metals), as related specifically to increased human exposure. For any organism, increased exposure to a given metal implies changes in the concentration and/or the speciation of the specific contaminant. Changes in concentration within various ecosystem components (atmosphere, water, biota) has been reported for some metals, related either to increased atmospheric deposition of the metal or to its increased mobilization in response to acidification. Even if total metal concentrations do not change, changes in metal speciation can be anticipated as a result of acidification, due to shifts in the hydrolysis, complexation, and adsorption equilibria.

For the metals (Hg, Cd, Pb and Al) judged to be of potential concern in the context of acidic deposition/health effects, the Biogeochemistry Working Group considered the following specific questions:

(1) importance of direct atmospheric deposition to a lake vs. inputs from watershed weathering;
(2) metal mobilization from the watershed and from sediments;
(3) influence of acidification on metal speciation (and hence bioavailability);
(4) effect of acidification on the microbial transformation of the metal;
(5) effect of acid water and increased atmospheric loading on water quality in water distribution systems.

The types of evidence considered in responding to these various questions are indicated in Table 2-1. As can be appreciated on examination of this table, our responses to questions (1), (2), (4) and (5) were straightforward. However, our approach to question (3), dealing with the influence of acidification on metal bioavailability, clearly merits some additional explanation.

In principle acidification could directly influence metal bioavailability by (i) shifting the equilibrium distribution of the dissolved metal complexes, both inorganic and organic, towards more available forms (e.g., the aquo ion, $M(H_2O)_n^{z+}$), or (ii) influencing metal-surface interactions at the

Table 2-1. Evidence considered in addressing the questions posed by the Biogeochemistry Working Group.

| Question | Type of Evidence |
|---|---|
| (1) | • whole-lake burdens of anthropogenic metals ($g/m^2$ or $g/m^2/yr$) in lakes receiving similar atmospheric inputs, but differing in [watershed/lake] area ratios |
| (2) | • metal concentration profiles in undisturbed soils |
| | • laboratory or field experiments in which soil columns have been subjected to simulated or real precipitation events, and the chemical composition of the percolate followed over time |
| | • regional surveys of soils, vegetation and/or surface waters in areas of relative geologic homogeneity, along a gradient in precipitation chemistry (increasing acidity) |
| | • metal concentration profiles in lake sediment cores |
| | • experimental acidification of streams, lakes |
| (3) | • thermodynamic calculations and experimental determinations of the equilibrium distribution of various metal forms, as a function of $[H^+]$ in the pH range 7 → 4 (assumption that bioavailability increases as the contribution of free aquo ion, $M(H_2O)^{z+}$, increases) |
| (4) | • laboratory studies of the kinetics of microbial transformations of metals, as a function of $[H^+]$ |
| | • regional surveys of metal transformation rates in situ, along a gradient in sediment / water chemistry (increasing acidity) |
| (5) | • regional surveys of water quality in public water supply systems and in private (dug or drilled) wells |
| | • evaluation of water quality changes occurring in water distribution systems with and without flows |

cell/medium interface. Depending upon the $pK_a$ values of the functional groups involved in metal binding and/or transport, the hydrogen ion may compete with the metal for binding sites at the cell surface and thus play a protective role analogous to the well-documented protection afforded by the hardness cations. A decrease in pH can thus be expected to affect both metal speciation in solution and biological sensitivity at the level of the cell surface. These two responses to acidification are inherently antagonistic.

Acidification may also indirectly influence metal bioavailability. For example, acidic deposition enhances sulfate reduction and denitrification in anaerobic habitats (Kelly et al., 1982). Of these, only sulfate reduction would have a significant effect on metal availability. Hydrogen sulfide, the product of bacterial sulfate reduction, forms extremely insoluble precipitates with many metals. This would effectively enhance migration of metals from the lower part of the water column to the sediment and reduce bioavailability of the metal, provided conditions remained anoxic. If aerobic conditions developed, the metal would be released by bacterial oxidation of the sulfide. With mercury, acidification may also influence metal bioavailability indirectly by affecting the microbial transformations of the metal.

Clearly there is no simple answer to the question "Does acidification affect metal bioavailability?". We have limited our response by considering only the biogeochemical aspects, i.e., the effect of acidification on metal speciation in solution. The Bioaccumulation Working Group has considered many of the other dimensions of the problem.

## 2.2 MERCURY

2.2.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering -- The atmospheric deposition rate of mercury to lakes generally exceeds watershed sources except under geologically unusual conditions (Hg-enriched bedrock). Indigenous sources of mercury in lakes sensitive to atmospheric deposition are generally small.

2.2.2 Mercury Mobilization from Watershed and Sediments -- Data for mercury are inconclusive. Almer et al. (1978) reviewed data for lake waters in Sweden showing an increase in the total concentration of mercury (unfiltered samples) as the lake pH decreased. In a subsequent publication, Dickson (1980) noted that this trend applied to lakes with different pH values, subjected to similar atmospheric loadings of mercury. However, this similarity of atmospheric loadings, a key point if the data are to be interpreted unambiguously in terms of acidification + mobilization from the watershed and/or sediments, could not be verified as the relevant atmospheric deposition data were not presented. Recently Lindqvist et al. (1984) concluded that reliable data on the concentration of mercury in lake water in Sweden are virtually non-existent, casting doubt on the reported trend towards increasing mercury concentrations with decreasing pH.

2.2.3 Influence of Acidification on Mercury Speciation -- Chemical equilibrium models can be used to calculate the distribution of mercury among a variety of representative inorganic and organic ligands at various pH values. Such simulations indicate that mercury speciation in inorganic media is very pH-sensitive over the pH range 7 → 4. At circumneutral pH values $Hg(OH)_2^o$ and $HgClOH^o$ are the dominant forms, whereas as the pH decreases $HgCl_2^o$ and $HgCl^+$

increase in importance; even at pH 4, however, less than 1% of the dissolved mercury is predicted to occur as the divalent aquo ion, $Hg(H_2O)_n^{+2}$. The biological significance of these speciation changes in unclear since the relative bioavailability of these different mercury forms is unknown.

The only experimental verification of these predictions performed under field conditions is that carried out in limnocorrals in the Experimental Lakes Area, Ontario, Canada (Jackson et al., 1980; Schindler et al., 1980). Lowering the pH within the enclosures from 6.7 → 5.7 → 5.1 decreased the ratio of dissolved to particulate mercury but did not affect the rate of mercury loss from the water column.

2.2.4 Effect of Acidification on Microbial Transformations of Mercury -- Mercury undergoes several microbially mediated transformations and has been studied more than any other heavy metals. Acidification affects these transformations, although the nature of the pH effects is largely unclear. Three transformations of mercury (methylation, demethylation, and reduction) are biologically mediated and will be discussed separately.

Methylation. Mercury methylation is predominantly a microbial process that exhibits greatest activity in surficial anoxic sediments. Although acidification has generally been thought to enhance methylation (Fagerstrom and Jernelov, 1972), recent evidence suggests that net methylation is inhibited with decreasing pH (J.W.M. Rudd, personal communication; Steffan and Winfrey, 1984).

This is in contrast to a growing amount of evidence showing an inverse relation between lake pH and mercury burdens in fish (Jernelov et al., 1975; Tsai et al., 1975; Brouzes et al., 1977; Jernelov, 1980; Tomlinson et al., 1980; Akielaszek and Haines, 1981; Stokes et al., 1983; Wiener, 1983; Lindqvist et al., 1984). Thus, the explanation for the increased mercury in fish must lie in factors other than enhanced biomethylation. Possible mechanisms include altered partitioning of methylmercury between sediments and water (Miller and Akagi, 1979), increased gill permeability (Pagenkopf, 1983), decreased trophic state of the lake (Hakanson, 1980), and decreased growth rate of fish (Jernelov, 1980).

A second contradiction appears when the effects of organic nutrients on methylation are examined. Increases in labile organic matter stimulate methylation (Bishop and Kirsh, 1972; Shin and Krenkel, 1976; Akagi et al., 1979; Furutani and Rudd, 1980; Wright and Hamilton, 1982), yet there is an inverse relation between the trophic state of a lake and mercury burdens in fish (D'Itri et al., 1971; Akielaszek and Haines, 1981; Lindqvist et al., 1984).

Demethylation. Methylmercury, once formed, may also be demethylated by microorganisms (Billen et al., 1975; Jernelov et al., 1975; Kudo et al., 1978; Jernelov and Martin, 1980). Little is known, however, concerning this process or the effect of acidification on it.

Mercury Ion Reduction. Mercury ion is readily reduced to volatile elemental mercury by mercury-resistant bacteria. This can result in release of mercury from an aquatic ecosystem. Steffan and Winfrey (1984) showed that volatilization was not inhibited by a decrease in pH in surface lake sediments. Little is known, however, about the process of mercury reduction in nature or the effects of acidification on it.

2.2.5 Effect of Acid Water and Increased Atmospheric Loading on Mercury Levels In Water Distribution Systems -- There are no sources of mercury in water distribution systems; consequently, concentrations of this metal are determined before entry into the distribution system, i.e., by the raw water quality. Human exposure to mercury via potable water supplies will be negligible because mercury concentrations are extremely low, even in low pH waters.

## 2.3 LEAD

2.3.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering -- Dillon and Evans (1982) demonstrated that total lake lead burdens ($g/m^2/y$ and $g/m^2$) varied only by a factor of 1.5 in lakes in the same geographical region but with various [watershed/lake] area ratios. This suggests little transport of lead from the drainage basin to the lake. Lead concentrations in precipitation are much greater than those in lakes and inlet streams to lakes (Norton et al., 1981b; Kahl and Norton, 1983). Lead concentrations in recent sediments greatly exceed those in older "pre-cultural" sediments (many authors). Therefore, lead inputs to the lake water column are primarily from direct atmospheric deposition to the lake surface; inputs via tributaries or inflow will be negligible, because lead is retained in terrestrial soils by various mechanisms.

2.3.2 Lead Mobilization from Watershed and Sediments -- Data for lead are conclusive: lead is clearly the least mobile of the four metals considered.

In laboratory and field experiments involving soil columns subjected to simulated or real precipitation events, lead was immobile, even when the columns were subjected to leaching with dilute acids (Fuller et al., 1976; Tyler, 1978). In a prolonged field study, Tyler (1981) quantified the amount of lead leached from the A-horizon of a podzolic spruce forest in southern Sweden; although some downward movement of lead occured, the metal was retained in the B horizon and not lost to the groundwater. For well-drained organic soils in North America, Hanson et al. (1982) reported that essentially all of the incoming lead in precipitation was retained in the organic soil layer, regardless of the pH of the incident precipitation (see also Smith and Siccama, 1981). Andren et al. (1975) developed a lead budget for the Walker Branch watershed near Oak Ridge, Tennessee; only 2 to 3% of the lead input was exported from the watershed by the stream.

The paleolimnological data available for lake sediments in Norway (Reuther et al., 1981), the northeastern U.S. (Hanson et al., 1982), Ontario (Dillon and Evans, 1982) and Quebec (Ouellet and Jones, 1983) all suggest that lead is strongly bound to lake sediments. The critical pH for release of lead from the sediment is less than the lowest observed water column pH values. Experimental confirmation of this immobility was recently provided by Davis et al. (1982), who subjected the upper strata from two sediment cores to acidification in the laboratory; significant lead release (> 5% of total concentration) occurred only at pH < 3.0. Kahl and Norton (1983) sampled sediments from lakes of pH 6-6.5 and subjected the undisturbed cores and the overlying water to acidification in the laboratory; significant release of lead was noted only at pH 4.0, with lead concentrations increasing from initial values of about 1 $\mu g\ \ell^{-1}$ to equilibrium values of 5-10 $\mu g\ \ell^{-1}$.

2.3.3 Influence of Acidification on Lead Speciation -- Computer simulations indicate that lead speciation should vary moderately in inorganic media over the pH range 7 → 4. At circumneutral pH values, the $PbCO_3^0$ and $PbOH^+$ complexes are predicted to exist in solution (> 50%), but below pH 6 the $Pb(H_2O)_n^{2+}$ aquo ion dominates (> 90%). Comparable calculations for media containing model organic ligands, similar to those used by Sposito (1981) to represent the acid-base and metal-complexing behavior of fulvic acid, do not reveal additional pH dependence of lead complexation in the presence of these ligands. However, experimental measurements of lead speciation in synthetic solutions containing humic acid do indicate an enhanced pH sensitivity in the presence of natural organic matter over the critical region 7 → 4 (Guy and Chakrabarti, 1976; O'Shea and Mancy, 1978); as would be anticipated, the degree of complexation decreased at the lower pH values.

2.3.4 Effect of Acidification on Microbial Transformations of Lead -- Several investigations have demonstrated biomethylation of lead (Wong et al., 1975; Huber and Schmidt, 1976; Thompson and Crerar, 1980), although recent studies have questioned the validity of these reports (Reisinger et al., 1981; Jarvie et al., 1983). Chemical methylation, however, is known to occur (Ahmad et al., 1980; Jarvie and Whitmore, 1981). Lead concentrations in fish were greater in naturally acidic than circumneutral clear-water lakes in northern Wisconsin (Wiener, 1983), although no data are available on the effect on pH on lead methylation. The reported lead burdens in fish or waters subject to cultural acidification do not appear to impose any human health hazard.

2.3.5 Effect of Acid Water and Increased Atmospheric Loading on Lead Levels in Water Distribution Systems -- Total lead concentrations in surface and groundwaters are typically less than 2 $\mu g\ \ell^{-1}$ (many surveys). In contrast, elevated lead concentrations (up to 200 $\mu g\ \ell^{-1}$) have been found in domestic water, allowed to equilibrate overnight in the household distribution system (Hultberg and Johansson, 1981; Norton et al., 1984). Thus the distribution system is dominant in determining lead levels. However, secondary effects of acidified water (e.g. sulfur compounds) may contribute to this mobilization (see Sections 4.3.1.1 and 4.3.1.3).

## 2.4 ALUMINUM

2.4.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering -- The atmospheric input of aluminum to lakes is small, relative to groundwater and surface water inputs derived from watershed weathering.

2.4.2 Aluminum Mobilization from Watershed and Sediments -- The increased geochemical mobility of aluminum in response to environmental acidification has been shown in numerous studies, including laboratory experiments involving the leaching of soil columns (Fuller et al., 1976), field studies of soil percolates

(Tyler, 1981), regional surveys of soil, groundwater and surface waters (Almer et al., 1978; Cronan and Schofield, 1979; Dickson, 1980; Johnson et al., 1981), the experimental acidification of lakes (Schindler and Turner, 1982) and streams (Hall et al., 1980), and the experimental acidification of sediments (Kahl and Norton, 1983).

2.4.3 Influence of Acidification on Aluminum Speciation -- Chemical equilibrium calculations indicate that the distribution of dissolved aluminum among its various inorganic complexes (hydroxo- and fluoro-complexes) should be highly sensitive to pH changes. At neutral pH values, the predominant species is $Al(OH)_4^-$, whereas at pH < 4 the trivalent aquo ion, $Al(H_2O)_n^{+3}$, dominates. In the critical pH range from 7 to 4 both mononuclear complexes ($AlOH^{+2}$, $Al(OH)_2^+$, $Al(OH)_3^o$, $AlF^{+2}$, $AlF_2^+$) and polynuclear complexes ($Al_n(OH)_n^{(3n-m)+}$) have been suggested as dissolved species. Introducing representative organic ligands into the model system increases the degree of aluminum complexation at pH ∿ 7, but has relatively little effect at pH 4. The pH sensitivity of aluminum speciation, already appreciable in the inorganic system, is thus predicted to be even more marked in the presence of organic ligands. The increased contribution of species such as $Al(H_2O)_n^{+3}$ and $AlOH^{+2}$ at the lower pH values would be expected to increase bioavailability (Driscoll et al., 1980; Heliwell et al., 1983), provided there is not an antagonistic interaction with the hydrogen ion at the cell surface.

2.4.4 Effect of Acidification on Microbial Transformations of Aluminum -- No biologically mediated transformations of aluminum have been reported.

2.4.5 Effect of Acid Water and Increased Atmospheric Loading on Aluminum Levels In Water Distribution Systems -- If no sources of aluminum are present in water distribution systems, concentrations of this metal will be determined by the initial water quality before into the distribution system. However, food preparation with aluminum containers/utensils may introduce large amounts of aluminum to food fluids; the resultant aluminum concentrations may be orders of magnitude greater than those in acidified surface and ground waters (see Sections 4.4.1.1 and 4.4.1.3).

## 2.5 CADMIUM

2.5.1 Importance of Direct Atmospheric Deposition to a Lake vs. Watershed Weathering -- Whole-lake burdens of anthropogenic cadmium, calculated on an areal basis for softwater lakes in southern Ontario (Evans et al., 1983), ranged over a factor of 2 and were unrelated to the ratio of watershed area to lake area, or to the water replenishment time of the lakes. The authors concluded that atmospheric deposition to the lakes' surfaces was an important input of cadmium to the lakes. Watershed contributions will also be important, however, particularly in areas receiving acidic deposition (see Section 2.5.2).

2.5.2 Cadmium Mobilization from Watershed and Sediments -- In laboratory experiments involving the leaching of soil columns with synthetic rain water (Fuller et al., 1976; Tyler, 1978), cadmium showed moderate mobility (similar to Zn; less than Al, greater than Pb; i.e., Al > Cd ∿ Zn > Pb). Field studies confirmed the sensitivity of cadmium mobility to minor changes in the pH of the soil percolate (Tyler, 1981); cadmium concentrations in soil leachates were positively correlated with hydrogen ion concentrations.

Total cadmium concentrations in lake waters in Sweden (unfiltered samples) increased in low pH lakes (Almer et al., 1978; Dickson, 1980), suggesting that cadmium mobility increases with acidification. However, as noted earlier for mercury (Section 2.2.2), atmospheric loadings of cadmium to these lakes were not reported. It is thus difficult to distinguish between "acidification + mobilization" and "acidification + concomitant increased atmospheric deposition" as possible explanations for the increase in cadmium concentrations at lower pH. Cadmium concentration profiles in sediment cores from several Norwegian lakes show a decrease in concentration in the most recent sediment strata (Reuther et al., 1981); these data have been interpreted as mobilization of cadmium (and zinc) from the surficial sediments in lakes where the pH of the overlying water column is presently below 4.9. Alternatively, it may be argued that there is less efficient sediment retention of the metal in low pH lakes (Norton et al., 1981; Evans et al., 1983). In either case, cadmium is clearly mobile in acidified environments.

2.5.3 Influence of Acidification on Cadmium Speciation -- Computer simulations, both for inorganic media and for synthetic solutions containing representative organic ligands, indicate that cadmium speciation should be pH - insensitive in the pH range 7-4; the free aquo ion, $Cd(H_2O)_n^{+2}$, is predicted to dominate throughout this pH range. Experimental measurements in natural waters (Engel et al., 1981), and in synthetic solutions with or without humic acid (Gardiner, 1974; Guy and Chakrabarti, 1976; O'Shea and Mancy, 1978), confirm this relative insensitivity of cadmium speciation to pH changes.

2.5.4 Effect of Acidification on Microbial Transformations of Cadmium -- No biologically mediated transformations of cadmium have been reported.

2.5.5 Effect of Acid Water and Increased Atmospheric Loading on Cadmium Levels in Water Distribution Systems -- Cadmium concentrations in surface and ground waters are typically $< 1$ µg $\ell^{-1}$, well below the recommended health limits (10 µg $\ell^{-1}$). Contact with water distribution systems will on occasionally elevate these values to 1-10 µg $\ell^{-1}$ (see Section 4.5.1.1). It is not known whether this is due to natural corrosion or to secondary chemical effects related to water acidification (e.g., sulfur compounds as contrasted with $CO_2$-charged water).

## 3. BIOACCUMULATION (J.G. Wiener, P.M. Stokes, and R. Sloan)

### 3.1 GENERAL INTRODUCTION

A toxin accumulates in an organism if the absorption rate exceeds the excretion rate. The bioaccumulation of trace contaminants secondary to acid deposition may result from ingestion of contaminated food, direct uptake from water, or both.

The Bioaccumulation Working Group identified mercury as the contaminant of primary concern with regard to human consumption of fish from acid-sensitive waters. For mercury, consumption of fish is considered the biological pathway of greatest importance to man. Dietary intake of methylmercury by man results almost entirely from consumption of fish and fish products (Clarkson, 1983). Accumulation of methylmercury in edible fish flesh during exposure of fish to mercury in water or food can be substantial (McKim et al., 1976; Scherer et al., 1975; Huckabee et al., 1978, 1979; Lodenius et al., 1983; Wren et al., 1983; Phillips et al., 1980). A substantial amount of the methylmercury in freshwaters may be contained within the biomass of the system (Clarkson and Baker, 1983).

Concentrations of certain other metals, such as cadmium and lead, in fish are inversely correlated with pH and related chemical characteristics of lakes; however, these metals tend to accumulate in tissues and organs not consumed by man (see Section 3.3). Aluminum can be acutely toxic to fish (via the gills) in acidic surface waters. Because of this acute toxicity, aluminum accumulation in fish is not significant from a human health perspective.

## 3.2 MERCURY

3.2.1 Accumulation of Mercury by Fish -- Most of the mercury present in edible fish flesh (generally > 80%) exists as methylmercury (Huckabee et al., 1979). Fish cannot methylate mercury in vivo (Huckabee et al., 1978), although methylation of mercury in the gastrointestinal tract has been documented (Rudd et al., 1980). Fish can accumulate methylmercury from water and food (Phillips and Buhler, 1978; Phillips and Gregory, 1979; Olson et al., 1973; Scherer et al., 1975; Rodgers and Qadri, 1982; Turner and Swick, 1983). The direct uptake of methylmercury from water occurs almost entirely across the gills (Olson et al., 1973). Total mercury concentrations in fish are often correlated with mercury levels in organisms of lower trophic levels (Hultberg and Hasselrot, 1981; Stokes et al., 1983; MacCrimmon et al., 1983), providing circumstantial evidence of the importance of dietary uptake in natural waters. Within a given fish community, piscivorous fish species generally contain the highest mercury concentrations in edible flesh (MacCrimmon et al., 1983; Lodenius et al., 1983; Wren et al., 1983; Phillips et al., 1980). A number of North American fish species, in which mercury concentrations in muscle have exceeded 1.0 μg $g^{-1}$ wet weight, are listed in Table 3-1.

Elimination of methylmercury by fish is very slow relative to the rate of uptake. Consequently, mercury concentrations in edible flesh of a fish species within a given water body generally increase with increasing age or body size (Huckabee et al., 1979; Phillips and Buhler, 1978).

3.2.2 Effects of Acidification on Mercury Bioaccumulation by Fish -- Accumulation of mercury by fish may increase as a result of acidification. Within a geographic area, mercury concentrations in edible fish flesh (of a given species and age) are often inversely related to lake pH or alkalinity (Table 3-2). Furthermore, total mercury concentrations in edible flesh of piscivorous fishes inhabiting low pH waters ($pH \leq 6.0$) frequently exceed 0.5 or 1.0 μg $g^{-1}$ wet weight. This pattern has been observed in remote lakes having no local anthropogenic sources of mercury (e.g., Bloomfield et al., 1980).

The relative importance of uptake from food and water in acidic lakes is not understood. The direct uptake of methylmercury across the gills of fish is apparently enhanced in waters having low pH and low calcium content (Drummond et al., 1974; Rodgers and Beamish, 1983). An increased dietary uptake of

TABLE 3-1.

North American fishes for which mercury concentrations exceeding 1.0 µg/g (wet weight) in edible muscle tissue have been reported.

| Species | Location | Reference |
|---|---|---|
| Largemouth bass, *Micropterus salmoides* | South Carolina reservoirs | Abernathy and Cumbie, 1977 |
| Smallmouth bass, *Micropterus dolomieui* | Tadenac Lake, Ontario | Wren et al., 1983 |
| Lake trout, *Salvelinus namaycush* | New York State lakes and rivers | Bloomfield et al., 1980 |
| | Tadenac Lake and other Precambrian Shield lakes | MacCrimmon et al., 1983 |
| Northern pike, *Esox lucius* | Northern Maine lakes | Akielaszek and Haines, 1981 |
| | Lake Hacksjon, Sweden | Gothberg, 1983 |
| | Tongue River reservoir, Montana | Phillips et al., 1980 |
| | Swedish lakes | Lindqvist et al., 1984 |
| | Tadenac Lake, Ontario | Wren et al., 1983 |
| Walleye, *Stizostedion vitreum vitreum* | Minnesota lakes | Glazer and Bohlander, 1978 |
| | Tongue River reservoir, Montana | Phillips et al., 1980 |
| | Minnesota lakes | Glazer and Bohlander, 1978 |
| | Southern Ontario lakes | Scheider et al., 1979 |
| | Michigan lakes | Kelly et al., 1975 |
| | Softwater lakes in northern Wisconsin | Wiener, 1983 |

TABLE 3-2.

Reported relationships between total mercury concentrations in fish and physicochemical characteristics of surface waters in North America.

| Location | pH range of waters studied (N)[a] | Matrix and species analyzed | Summary of findings | Reference |
|---|---|---|---|---|
| Lakes near Muskoka, Ontario | 5.1-7.5 (14) | Whole, yearling (1+ age) yellow perch | Mercury concentration in yellow perch was significantly ($P < 0.05$) correlated with epilimnetic pH ($r$ = 0.63), aluminum concentration in epilimnetic water ($r$ = 0.65), and watershed area/lake volume ratio ($r$ = 0.92). | Suns et al., 1980 |
| Lakes in south-central Ontario | Not reported (52) | Axial muscle tissue of walleye | Mercury concentrations in walleyes of a given length group were greater in fish from low alkalinity lakes (≤300 μeq/L) than in fish from high alkalinity lakes (>300 μeq/L). Mean mercury concentrations of large (>48 cm) walleyes in low alkalinity lakes equaled or exceeded 1.0 μg/g. | Scheider et al., 1979 |
| Lakes in north-central Wisconsin | 5.6-7.0 (5) | Axial muscle tissue of walleye | Mercury concentrations were significantly ($P < 0.01$) greater in 4-, 5-, and 7-year-old fish from two naturally acidic lakes (pH 5.6 and 6.0) than in fish of the same age from three circumneutral (pH 6.9-7.0) lakes. Mercury concentrations in about one-third of the fish from the naturally acidic lakes exceeded 1.0 μg/g wet weight. | Wiener, 1983 |
| Lakes in the Adirondack Mountains, New York | 4.9-7.2 (15) | Whole brook trout | The mean mercury concentration, adjusted for length, was greater ($P < 0.01$) in fish from acid lakes (pH ≤ 5.0) than in fish from lakes with pH > 5.0. None of the 200 brook trout analyzed contained mercury concentrations exceeding 1.0 μg/g. | Sloan and Schofield, 1983 |
| Lakes and bays in south-central Ontario | 5.6-8.4 (16) | Axial muscle tissue of pumpkinseed | Mercury concentration in pumpkinseed was significantly ($P < 0.05$) correlated with pH ($r$ = -0.66), conductivity ($r$ = -0.64), alkalinity ($r$ = -0.63), waterborne calcium ($r$ = -0.62), and lake volume ($r$ = -0.55). A multiple regression equation with four independent variables (percent littoral zone, pH, maximum depth, and lake volume) accounted for 93% of the variation in mercury content of fish. The maximum mercury concentration reported was 0.54 μg/g. | Wren and MacCrimmon, 1983 |
| Headwater lakes in the Sault Ste. Marie District, Ontario | 5.8-6.9[b] | Carcasses of brook trout, with gonads and stomach contents removed | Mercury concentration in fish was significantly ($P < 0.05$) correlated with sodium concentration in water ($r$ = -0.71). The maximum mercury concentration observed was 0.79 μg/g wet weight. | Kelso et al., 1982 |
| Headwater lakes in the Sault Ste. Marie District, Ontario | 5.2-7.0[b] | Carcasses of white sucker, with gonads and stomach contents removed | Mercury concentrations in fish were significantly ($P < 0.05$) correlated with lake area ($r$ = 0.45) and waterborne calcium ($r$ = -0.34). The maximum mercury concentration observed was 0.22 μg/g wet weight. | Kelso et al., 1982 |

---

[a] N is the number of waters studied.
[b] N was not given.

methylmercury could also occur in acidic waters, due to increased mercury accumulation by organisms in lower trophic levels and subsequent passage through the food chain (Stokes et al., 1983).

Elevated mercury levels in fish inhabiting naturally acidic or culturally acidified waters have been observed in Scandinavia, Ontario, Wisconsin, and the Adirondack region of New York (Hakanson, 1980; Hultberg and Hasselrot, 1981; Bloomfield et al., 1980; Scheider et al., 1979; Wiener, 1983; Lindqvist et al., 1984; Jernelov, 1980; Sloan and Schofield, 1983). Multiple correlation analysis of Swedish data by Jernelov (1980) indicated that lake pH explained about 50% of the variation in mercury levels in 1-kilogram northern pike. Hultberg and Hasselrot (1981) also evaluated factors affecting mercury contamination of fish in 152 Swedish lakes. The mercury concentration in muscle of northern pike was inversely correlated with lake pH, and it was estimated that a one unit decrease in pH would increase mercury concentration in the muscle tissue of pike by 0.14 $\mu g\ g^{-1}$.

Several mechanisms have been proposed (Jernelov, 1980; Wood, 1980; Haines, 1981) as potential explanations of the relation between the acidity of surface waters and the bioaccumulation of mercury.

(1) Tomlinson et al. (1980) hypothesized that acidic precipitation may scavenge mercury from the atmosphere more effectively than nonacidic precipitation. This hypothesis does not explain the large differences in the mercury content of fish from acidic and nearby circumneutral lakes (e.g., Wiener, 1983).

(2) Jernelov (1980) proposed that formation of monomethylmercury by microorganisms is pH-dependent, with the maximum rate occurring at pH 6.0. Methylation is higher at pH 5.0-7.0 than at higher pH values. In addition, it has been hypothesized that low pH levels ($pH \leq 6.5$) enhance the formation of monomethylmercury, rather than dimethylmercury, which is volatile and released from the aquatic system to the atmosphere (Fagerstrom and Jernelov, 1972). Thus, at these lower pH levels more monomethylmercury -- the form most readily assimilated by fish -- would presumably be present, and bioaccumulation would be enhanced. However, this proposed mechanism is not supported by recent investigations. In situ studies by J. W. M. Rudd (pers. comm.) and laboratory studies by Steffan and Winfrey (1984) have shown that decreases in pH (below pH 6.5) slow the net rate of monomethylation of $^{203}Hg$ added to surficial sediments. Thus, the net rate of monomethylmercury formation is probably not greater at low pH. Furthermore, Miller and Akagi (1979) showed that methylmercury production was not pH-dependent in three types of sediment across the pH range 5.0 - 7.5.

(3) Inorganic mercury is more soluble in acidic media (under aerobic conditions) and more potentially available for methylation reactions. Retention of mercury in the water column is thus enhanced at low pH (Jackson et al., 1980), increasing the exposure of fish to mercury. For example, Miller and Akagi (1979) performed laboratory experiments on three sediment types

and showed that the equilibrium partition coefficient $[\mathrm{MeHg}]_{\mathrm{sed}}/[\mathrm{MeHg}]_{\mathrm{water}}$ decreased between pH 7 and 6, and between pH 6 and 5 for each of the sediment types. They hypothesized that the effect of pH on methylmercury partitioning between water and sediment was responsible for the high bioavailability of mercury in acidic waters. However, their experiments were conducted with very high mercury concentrations.

(4) Tomlinson et al. (1980) proposed two potential mechanisms leading to increased mercury contamination of biota in acid waters. First, reductions in biomass in acidic lakes would increase mercury availability per unit weight to the remaining biomass, resulting in higher mercury burdens in biota. For example, a decrease in the growth rate of fish in an acidified lake would presumably be accompanied by increased mercury concentration in tissues -- the accumulated mercury being contained in a smaller biomass. Second, as a lake acidifies, the forage base for piscivorous fishes is altered such that they feed on larger fish with higher mercury content. Empirical support for these hypotheses is lacking.

There are presently no definitive experimental data showing a cause and effect relationship between acidification and increased mercury accumulation by aquatic biota. Experimental work on biogeochemical factors enhancing mercury accumulation by fish in dilute, acidic waters is therefore needed. This need is especially urgent in the United States, where few data are available.

3.2.3 Effects of Productivity on Mercury Uptake by Fish -- Fish in oligotrophic lakes often have higher mercury concentrations than fish in more productive systems (D'Itri et al., 1971; Hakanson, 1974). Two potential explanations for this have been offered, one relating to mercury methylation and the other relating to mercury complexation. Akielaszek and Haines (1981) hypothesized that methylation of mercury is enhanced in oligotrophic lakes. Hakanson (1980) cited several studies showing a relation between productivity and suspended organic matter, which provides sites for complexation of mercury, thus reducing the fraction of mercury in the water column available to fish.

In contrast, organic enrichment of mercury-polluted systems appears to increase mercury uptake by fish. For example, addition of organic matter such as wood chips, moss, or leaves to enclosures in mercury-polluted Clay Lake in the Wabigoon River system in Ontario increased the mercury levels in fish (Rudd et al., 1983). Even in the absence of gross mercury pollution, the addition of organic matter to limnocorrals in the Southern Indian Lake reservoir in northern Manitoba increased both methylation rates and mercury concentrations in fish (Bodaly et al., 1984). These results were interpreted as the stimulation of microbial or chemical methylation of mercury, where the quantity of mercury present is presumably not the limiting factor. Rather, the organic matter available for methylation may be limiting. This interpretation is supported by the observations of H. Hultberg (pers. comm.) on fish and zooplankton and by P. M. Stokes (unpublished data) on filamentous algae. Field surveys by both Hultberg and Stokes revealed a positive correlation between dissolved organic carbon in oligotrophic lakes and mercury concentration in biota.

### 3.2.4 Effects of Chemical Neutralization of Lakes on Mercury Contamination of Fish

-- The mercury content of fish has been statistically related to pH, waterborne calcium, alkalinity, and other physicochemical variables (Table 3-2). The effect of liming (the addition of $CaCO_3$, $Ca(OH)_2$ or dolomite limestone) on mercury levels in fish should therefore be evaluated. Decreases in the mercury content of fish after liming have been reported (Suns et al., 1980). However, mercury levels in fish did not decrease after liming in many Swedish lakes (H. Hultberg, pers. comm.). Without a better understanding of the mechanisms by which aqueous chemistry affects the availability of mercury to fish, predictions will remain speculative.

### 3.3 OTHER METALS

Certain other metals exhibit elevated concentrations in acid waters and could potentially accumulate in fish. For example, body burdens of cadmium and lead are inversely correlated with pH and related chemical characteristics of lakes in some areas (Kelso et al., 1982; Wiener, 1983; U.S./Canada, 1983; Kelso and Gunn, 1984). Increasing accumulation of inorganic lead with decreasing pH has also been shown in laboratory studies (Merlini and Pozzi, 1977; Hodson et al., 1978b). However, concentrations of cadmium and lead in edible fish flesh (axial muscle tissue) do not increase greatly with increasing exposure concentration (Holcombe et al., 1976; Jaakkola et al., 1972; Bollingberg and Johansen, 1979; Sangalang and Freeman, 1979; Westernhagen et al., 1980). Rather, lead and cadmium tend to accumulate in tissues and organs that are not generally consumed by man, such as the kidney, liver, and bone (Holcombe et al., 1976; Patterson and Settle, 1977; Hodson et al., 1978a; Kumada et al., 1980; Fjerdingstad and Nilssen, 1983).

The extent to which aluminum is accumulated by fish has been little studied. It is, however, known that the mode of aluminum toxicity to fish is external (on the gills), not internal (Muniz and Leivestad, 1980). Aluminum can be toxic to fish at waterborne concentrations as low as 0.1-0.2 mg $\ell^{-1}$ (Schofield and Trojnar, 1980; Muniz and Leivestad, 1980; Baker and Schofield, 1982), which is within the range of concentrations measured in many acidic surface waters. Buergel and Soltero (1983) reported aluminum concentrations in muscle tissue of rainbow trout (*Salmo gairdneri*) between 0.003 - 0.18 μg $g^{-1}$ dry weight. Therefore, based on limited results, aluminum apparently has little tendency to accumulate in edible fish tissue.

## 4. INDIRECT HEALTH EFFECTS

(T.W. Clarkson, D.A. Bennett, R.A. Goyer, S. Joshi, M. McDonald, R.G. Miller, P.D. Moskowitz, G. Nordberg, H. Olem, M. Piscator)

### 4.1 GENERAL INTRODUCTION

The most conclusive evidence of cause and effect relationship between an environmental factor and human disease is obtained from observations made on human populations. However, the indirect health effects from the acidic

---

Footnote: Parts of this section regarding mercury, lead and drinking water systems were extracted from Chapter E-6 of the Critical Assessment Document (EPA-600/8-83-016B). Acknowledgement: T.W. Clarkson, J.P. Baker, and W.E. Sharpe.

deposition phenomenon do not provide opportunity for these types of observations. Numerous factors contribute to this restriction, the nature of the contaminants involved and the types of illness that might be expected to be observed.

Of particular interest are contaminants such as lead, cadmium, mercury and other trace elements. Virtually everyone in the general population has already accumulated some level of these substances in body tissues; further, there is nearly continuous exposure to these contaminants, although in some instances very small, from multiple sources, and are both natural and anthropogenic. All trace contaminants have known toxic effects on man. But with few exceptions (heme effects of lead) the expected health effects include common ailments experienced by the general population such as behavioral and neurologic diseases, renal disease, reproductive disorders, neoplasia and other common disorders. For each of these toxicants, expected dose-response relationships are established and surveillance of human populations permits protection from expected health effects by elimination of likely sources.

The potential contribution of acidic deposition to human disease lies in the role it may have in increasing human exposure to trace substances, thereby making control of human exposure more difficult (contamination of water supplies), or even so restrictive of life style as to impose other undesirable effects (contamination of food supplies). If such extremes are permitted it may be exceedingly costly both monetarily and healthwise to reverse such effects. Therefore, investigation is warranted of the contributions of the acidic deposition to human exposure to these contaminants.

The metals and other contaminants selected for discussion were chosen because they may present a potential human health hazard. The toxicology of these substances - mercury, lead, cadmium, aluminum, asbestos and nitrates - was discussed by the working group in some detail. Mercury, in the form of methylmercury, is toxic to the central nervous system in both the human adult and fetus. Lead also can produce serious effects in the nervous system, as well as in the hemopoietic system and kidneys. Cadmium has an uncommon capacity to accumulate in the human body virtually over the entire human lifespan with the kidney as the major site of damage for cadmium. Aluminum continues to be implicated in degenerative diseases of the central nervous system. Asbestos, when inhaled, is a potent carcinogen. Nitrates are known to cause methemoglobinemia in infants. The Health Effects Working Group discussions include the effects of these agents at different stages of the human life cycle. The potential for prenatal damage was noted for methylmercury and for lead. Cadmium may affect the function of the placenta.

These health effects will be described in further detail in this report. The Human Health Effects Working Group recognized the potential for direct health effects, however, believes that the main interest at this time is in the potential for acidic deposition to mobilize these substances and thereby increase human exposure. If future research identifies large increases in human exposure due to acidic deposition, then the subject of health effects including dose-effect and dose-response relationships will come sharply into focus.

The postdepositional pathways of human exposure are principally intake from food and drinking water. Hemodialysis is an exception and is of interest with regard to exposure to aluminum. The mechanisms of mobilization of toxic substances into food and drinking water are discussed by the Biogeochemistry and Bioaccumulation working groups and will only be discussed here, where appropriate.

Food. Mobilization of toxic substances into human food might occur via the aquatic food chain. The accumulation of methylmercury in edible tissues of fish and the relationship to acid deposition is discussed by other working groups (see Sections 2. and 3.). Information is sparse on mobilization into terrestrial food chains.

Human exposure might be affected by the state of combination of toxic substances in food. For example, cadmium in certain edible animal tissues may be bound to metallothionein and in this form be distributed within the human body in a different way from cadmium not bound to metallothionein.

Drinking Water. An understanding of the modes of hydrologic interactions between acid deposition and various types of water supply systems is essential to assessing the potential indirect health effects to users of drinking water obtained from each type of system. In addition, the physical facilities used to store, treat, and distribute water are of primary importance, as are the chemical methods used to treat water prior to use. Principal water sources in continental North America are usually either surface or groundwater, with other sources such as direct use of precipitation of much lesser importance. Health risk is directly related to the source of drinking water.

Health risk in drinking water supplies is also closely related to the management of the drinking water supply. Risks are generally greater the smaller the water supply, with small privately owned water systems serving a single dwelling at greatest risk. These systems typically do not routinely monitor water quality nor do they provide even rudimentary water treatment. Data on the impacts of atmospheric deposition on drinking water quality are extremely scarce; however, by using available information on the impacts to surface water aquatic ecosystems, we may assess impacts.

Precipitation was directly used for water supply in North America from very early times. Its use is still common where there are no other water supply alternatives. In the equatorial regions of the world, island communities still rely heavily on rainwater cisterns to supply their freshwater needs, and this method of water supply is being seriously considered as appropriate technology to supply drinking water for the developing countries of the world.

Since, in most systems, precipitation is used directly with no treatment, the quality of precipitation and the amount of dry deposition on the catchment between precipitation events are of paramount importance to the quality of drinking water at the user's tap. The major impacts are twofold: (1) direct deposition of atmospheric pollutants such as lead and copper may occur, and (2) the acid components of atmospheric deposition may cause increased corrosion of metallic plumbing system components, as well as the release of asbestos fibers from some types of roofing material into cistern water supplies.

Surface water supplies may be impacted by acidic deposition in two ways: (1) the quality of the source water may be impaired, and (2) increases in the corrosivity of the water could increase corrosion of metal plumbing systems. Corrosivity is probably the most significant potential concern related to atmospheric deposition on surface water supplies. The corrosivity of the dilute water often used for surface water supplies in the northeastern United States is mostly controlled by hydrogen ion concentration. As the hydrogen ion concentration increases so does the corrosivity of the water.

Corrosivity in surface water supplies has been widely reported, and its impacts are well documented. Where lead water distribution pipes are in use, clinical lead poisoning of children has been reported as a consequence of corrosive drinking water conveyance. A notable example of such a problem is Boston, Massachusetts. Less well known is the case of Mahanoy City, PA (Kuntz, 1983). A case of copper toxicity from a corroded water fountain has also been reported by Semple et al., (1960). Where pipes are of other metals such as copper, iron, or galvanized steel the corrosion products of copper, lead, iron, zinc, and cadmium can affect water quality. Where asbestos/cement piping is used in central water distribution systems, asbestos fibers may appear in the water. Because corrosion can lead to elevated concentrations of toxic contaminants in drinking water, the U.S. Environmental Protection Agency (1979a) has recommended that all drinking water supplies be noncorrosive and that a minimum pH of 6.5 be maintained. Taylor et al., (1984) in a study of acidic deposition and drinking water quality in the eastern United States, found that generally when the pH of raw water was lower than 6.5 the drinking water standards for trace contaminants (U.S. EPA, 1979b) were not met.

Acidification of groundwater as a consequence of atmospheric deposition has been reported in Sweden by Hultberg and Wenblad (1980), such changes have not as yet been well documented in North America. The need for documentation is greatest with respect to groundwater impacts from atmospheric deposition. Although additional work is indicated, preliminary information seems to indicate that adverse impacts to drinking water quality are possible in water supplies using shallow groundwater in areas edaphically and geologically sensitive to atmospheric deposition.

## 4.2 MERCURY

### 4.2.1 Environmental Exposure and Uptake by Humans

4.2.1.1 Concentrations in Water -- Bodies of fresh water having no known source of mercury generally yield values less than 200 ng $\ell^{-1}$. Most values fall in the range of 10 to 40 ng $\ell^{-1}$ and drinking water usually has values less than 30 ng $\ell^{-1}$ (WHO, 1976).

Few reports exist on the speciation of mercury in water, probably because of analytical difficulties. A recent review by McLean et al., (1980) found that methylmercury accounted for a small fraction of the total approximately in the order of 1 percent. However, a more recent report by Kudo et al., (1982) found that methylmercury accounted for about 30 percent of total mercury in samples taken from Canadian and Japanese rivers. Mercuric mercury ($Hg^{2+}$) accounted for about 50 percent.

Two important conclusions may be drawn from these data. First, that precipitation is an important source of mercury to fresh water, and second, that mercury in drinking water offers no health threat (see Section 2.2.1). Concentrations on the order of a few hundred nanograms per liter would result in a negligible intake of mercury on the assumed intake of two liters per day (U.S. EPA, 1980a). This intake, less than 2 µg $day^{-1}$, is well below the advised maximum safe intake of 30 µg Hg $day^{-1}$ (WHO, 1972); thus, additional mobilization of mercury into water by acidic deposition should not pose a health threat in terms of contaminated drinking water.

4.2.1.2 Concentrations in Aquatic Biota -- The concentration of methylmercury in fish tissue is of special interest in terms of human exposure. Bioaccumulation of methylmercury in fish is the principal if not the sole source of human exposure, barring episodes of accidental discharge or misuse of man-made methylmercury compounds. Thus, factors that affect concentrations of methylmercury in edible fish tissue are of considerable importance in assessing potential human health risks from this form of mercury (see Sections 2.2 and 3.2).

4.2.1.3 Human Uptake and Risk Assessment -- Dietary intake accounts for the greatest fraction of total mercury intake by man (Table 4-1). Methyl mercury intake is exclusively from the diet and almost entirely from fish and fish products. The evidence comes from dietary studies showing close correlation of blood levels with fish consumption (Swedish Expert Group, 1971) and from large-scale analyses of food items in several countries, indicating that significant concentrations of methylmercury are found only in fish and fish products (U.S. EPA, 1980a).

Based on data from the National Marine Fisheries, Cordle et al., (1979) have reported a ranking of species of fish according to annual consumption in the United States (Table 4-2). The table clearly demonstrates that oceanic fish, especially tuna, account for the major amount consumed. However, when consumption is expressed according to the consumer use, a different picture emerges. On this basis, freshwater fishes dominate the rankings, with northern pike consumed at 17.4 g $day^{-1}$, followed by trout (freshwater) at 12.3 g $day^{-1}$, bass (freshwater) and catfish at 12.1 g $day^{-1}$. The highest per user consumptions of seafood are crabs and lobster at 10.6 g $day^{-1}$, with tuna down to 6.1 g $day^{-1}$.

The highest average mercury concentrations are also found in freshwater fish - northern pike at 0.61 µg Hg $g^{-1}$ wet weight and trout at 0.42 µg Hg $g^{-1}$ wet weight. Thus a northern pike consumer would have a daily average intake of methylmercury of 10.4 µg exclusively from northern pike, and a trout consumer would have had an average intake of 5.2 µg Hg. These average values are well below the recommended maximum safe intake of 30 µg Hg $day^{-1}$.

TABLE 4-1. ESTIMATES OF AVERAGE AND MAXIMUM DAILY INTAKES OF MERCURY BY THE "70 kg MAN" IN THE UNITED STATES POPULATION (ADAPTED FROM U.S. EPA 1980a)

| Media | Mercury intake µg $day^{-1}$ 70 $kg^{-1}$ (average) | Predominate form |
|---|---|---|
| Air | 0.3 | $Hg^o$ |
| Water | 0.1 | $Hg^{2+}$ |
| Food | 3.0 | $CH_3Hg^+$ |

TABLE 4-2. ESTIMATED FISH AND SHELLFISH CONSUMPTION IN THE UNITED STATES STATES RANKED ACCORDING TO ANNUAL CONSUMPTION FOR THE PERIOD SEPTEMBER 1973 TO AUGUST 1974 (ADAPTED FROM U.S. EPA 1980a AND CORDLE ET AL. 1979).

| | Rank | Amount $10^6$ lb $y^{-1}$ | Percent of total by weight | Number of actual users (millions) | Mean Amount per user, (g $day^{-1}$) | Average conc. of mercury µg Hg $g^{-1}$[a] |
|---|---|---|---|---|---|---|
| Total | | 2957 | 100.0 | 197.0 | 18.7 | |
| Tuna (mainly canned) | 1 | 634 | 21.4 | 130.0 | 6.1 | 0.14[b]<br>0.27<br>0.35 |
| Unclassified (mainly breaded, including fish sticks) | 2 | 542 | 18.4 | 68.0 | 10.0 | c |
| Shrimp | 3 | 301 | 10.2 | 45.0 | 8.3 | 0.05 |
| Ocean Perch[d] | 4 | 149 | 5.0 | 19.0 | 9.7 | 0.13 |
| Flounder | 5 | 144 | 4.9 | 31.0 | 8.6 | 0.10 |
| Clams | 6 | 113 | 3.8 | 18.0 | 7.6 | 0.05 |
| Crabs/lobsters | 7 | 110 | 3.7 | 13.0 | 10.6 | 0.07-0.14[e] |
| Salmon | 8 | 101 | 3.4 | 19.0 | 6.7 | 0.08 |
| Oysters/scallops | 9 | 88 | 3.0 | 14.0 | 7.8 | 0.03 |
| Trout[f] | 9 | 88 | 3.0 | 9.0 | 12.3 | 0.42 |
| Cod[d] | 11 | 78 | 2.7 | 12.0 | 8.1 | 0.14 |
| Bass[f] | 12 | 73 | 2.5 | 7.6 | 12.0 | c |
| Catfish[f] | 12 | 73 | 2.5 | 7.5 | 12.1 | 0.15 |
| Haddock[d] | 12 | 73 | 2.5 | 11.0 | 8.6 | 0.11 |
| Pollock[d] | 15 | 60 | 2.0 | 11.0 | 6.8 | 0.14 |
| Herring/smelt | | | | | | |
| Sardines | 16 | 54 | 1.8 | 10.0 | 6.7 | 0.02 |
| Pike[f] | 17 | 35 | 1.2 | 2.5 | 17.4 | 0.61 |
| Halibut[d] | 18 | 32 | 1.1 | 5.0 | 8.0 | 0.19-0.53 |
| Snapper | 18 | 32 | 1.1 | 4.3 | 9.3 | 0.45-369 |
| Whiting | 20 | 25 | 0.9 | 3.2 | 9.7 | c |
| All other Classified | | 152 | 5.1 | | | c |

[a] U.S. Chamber of Commerce (1978).

[b] Average values for skipjack, yellow fin, and white tuna, respectively.

[c] Data not available.

[d] Mainly imports.

[e] King crab - all others, respectively.

[f] Fresh Water.

The National Marine Fisheries Service developed an extensive data bank on fish consumption by individuals according to fish species (U.S. Department of Commerce, 1978). These data were based on a Diary Panel Survey of approximately 25,000 individuals chosen to be representative of the U.S. population. These data, along with additional information on mercury concentration of edible tissues of various fish species, allowed a calculation of the number of individuals who would be expected to exceed the maximum safe daily intake of 30 µg. It was calculated that 47 of the 25,000 individuals would exceed this limit by a small margin from consumption of fish and that 23 of these were consumers mainly of freshwater fish. According to calculations by Nordberg and Strangert (Figure 4-1) the risk of parathesia (see Section 4.2.2.2) at this level of intake will be small -- on the order of 0.3 percent.

### 4.2.2 Effects on Human Health

4.2.2.1 Metabolism in Humans -- The U.S. EPA (1980a) has reviewed information on uptake, distribution, and excretion of methylmercury in man. Methyl mercury is almost completely absorbed from the diet ( i.e., between 90 to 100 percent of the amount ingested is absorbed). After absorption in the gastrointestinal tract, methylmercury passes into the bloodstream and is distributed to all organs in the body. Approximately 5 percent of the absorbed dose goes to the blood compartment and 10 percent to the brain -- the target organ for toxic effects.

After the initial distribution is completed, usually a matter of a few days in man, the brain to blood concentration ratio is roughly constant, having values between 5:1 to 10:1. Methyl mercury is accumulated in growing hair. At the time of formation of the head hair, the ratio of the concentration of mercury in hair to the simultaneous concentration in blood is roughly constant and has an average value of about 250:1. Once incorporated into the hair, the mercury concentration remains constant. Because human head hair grows about 1 centimeter per month, analyzing centimeter segments of hair can recapitulate average monthly concentrations of methylmercury in blood. Measurements of mercury in samples of blood or hair are now routinely used to assess the body burden of methylmercury in humans and as an indicator of brain concentrations.

Methyl mercury is excreted from the body mainly in feces. Before excretion in the feces, methylmercury is converted into inorganic mercury. The site of this conversion is not known, but microflora in the lower gut are known to possess this capability. The rate of elimination from the body is directly proportional to the body burden. It is well described by a single exponential function characterized by a half-time of about 70 days. An important conclusion from this kinetic information is that it will take about one year for humans to attain a state of balance,i.e., to attain maximum steady body burden of methylmercury for any given daily intake in the diet. After cessation of given exposure, it will take one year for the body burden to fall to pre-exposure levels. Thus, dietary intake of methylmercury from fish should be evaluated over a matter of months. Intake on any one single day does not normally make an important contribution to the overall body burden.

Considerable individual differences exist in biological half-times in man although the average value is 70 days with a range of 30 to 180 days. The distribution is bimodal with 90 percent of the values distributed about an

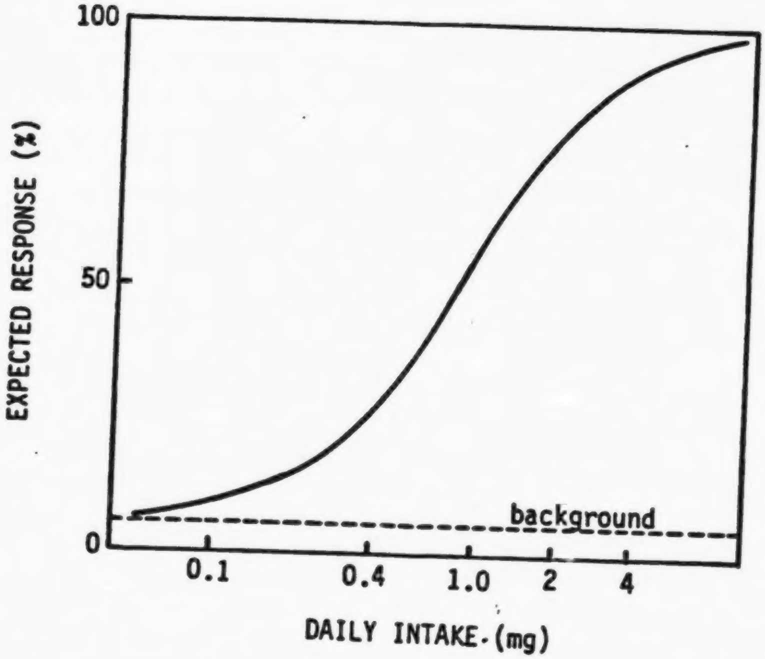

Figure 4-1. The calculated relationship between frequency of paresthesia in adults and long-term average daily intake of methyl mercury. The calculations were performed by Nordberg and Strangert (1978). The broken line is the estimated background frequency of paresthesia in the population. Data are taken from publications on the Iraqi outbreak of methyl mercury poisoning (Bakir et al. 1973; Shahristani & Shihab 1974).

average value of about 65 days and 10 percent distributed about an average value of 120 days. The reasons for this wide range of biological half-times are not known, except that lactating women have a short half-time averaging about 40 days.

Methylmercury readily crosses the placental barrier and enters the fetus. It distributes to all tissues in the fetus, including the fetal brain, which is the principal target for prenatal toxicity of methylmercury. Levels of methylmercury in cord blood are usually higher than the maternal blood concentrations.

Methylmercury is secreted in milk. Thus body burdens of methylmercury acquired by the infant before birth may be maintained by breast feeding if the nursing mother continues to be exposed to methylmercury.

The rate of elimination of methylmercury from the human fetus and suckling infant is not known. Experiments on animals indicate that elimination in suckling animals is much slower than in adults. The adult rate of excretion appears to commence at the end of the suckling period.

In brief, methylmercury accumulates in the human body over a period of about one year. Blood and hair analyses may be used as indicators of human absorption of mercury. In assessing hazard to human health, chronic exposure over weeks or months is important.

4.2.2.2 Effects and Dose-Response in Humans -- Methyl mercury damages primarily the human central nervous system. When ingested in sufficient amounts, methylmercury destroys neuronal cells in certain areas of the brain, the cerebellum and the visual cortex, resulting in permanent loss of function. Symptoms of damage include loss of sensation, constriction of the visual fields, and impairment of hearing. Coordination functions of the brain are also damaged, leading to ataxia and dysarthria. Severest damage causes mental incapacitation, coma, and death. The mildest and earliest effect in adults is usually a complaint of paresthesia, an unusual sensation in the extremities and around the mouth. In the Japanese population poisoned by methylmercury from contaminated fish, paresthesia was usually permanent. In the Iraqi population, paresthesia was frequently reported to be transient. This population had consumed homemade bread from wheat contaminated with a methylmercury fungicide.

The effects on the fetal brain differ qualitatively from those seen in adults. Methyl mercury interferes with the normal growing processes of the brain. It inhibits migration of neuronal cells to their final destination, thus affecting the brain's architecture. This damage manifests itself as diminished head size (microcephaly) and gross neurological manifestations such as cerebral palsy. The mildest effects are delayed achievement of developmental milestones in children and the presence of abnormal reflexes and mild seizures.

Brain concentrations associated with the onset of human methylmercury poisoning are in the range of 1 to 5 μg Hg g$^{-1}$ wet tissue. Blood concentrations for the onset of the mildest effects have been established to be between 200 and 500 ng Hg ml$^{-1}$ whole blood. Corresponding hair concentrations would be 50 to 125 μg Hg g$^{-1}$ hair (Table 4-3). The chronic daily intake of methylmercury that would lead to a maximum blood level of 200 ng ml$^{-1}$ has been established to be

TABLE 4-3. THE CONCENTRATIONS OF TOTAL MERCURY IN INDICATOR MEDIA AND METHYL MERCURY ASSOCIATED WITH THE EARLIEST EFFECTS IN THE MOST SENSITIVE GROUP IN THE ADULT POPULATION[a] (ADAPTED FROM WHO 1976)

| Concentrations in indicator media | | |
|---|---|---|
| Blood ($ng\ ml^{-1}$) | Hair ($\mu g\ g^{-1}$) | Equivalent long-term daily intake ($\mu g\ kg^{-1}$ body weight) |
| 200 to 500 | 50 to 125 | 3 to 7 |

[a] The risk of the earliest effects can be expected to be between 3 to 8 percent, i.e., between 3 to 8 percent of a population having blood levels in the range 200 to 500 $mg\ ml^{-1}$, or hair levels between 50 to 125 $\mu g\ g^{-1}$ would be expected to be affected (for further details, see text).

300 μg Hg. However the blood level in the mother associated with the earliest damage to the fetus has not yet been determined.

A major epidemiological study was carried out in Northwestern Quebec on Cree Indians exposed to methylmercury in freshwater fish (Methyl Mercury Study Group, 1980). The authors claim to find an association in men over age 30 and women over age 40 of a set of neurological abnormalities and the estimated exposure to methylmercury. However, it should be pointed out that this association has been seen by only four of seven observers who reviewed video taped recordings of the neurological screening tests. The severity of these neurological abnormalities was assessed by neurologists as mild or questionable. It was not possible to estimate any threshold body burden or hair levels because this population had been exposed possibly for most of their lives; therefore peak values in previous years are unknown. However, observations on this population over several years indicate that maximum blood concentrations are below 600 ng $ml^{-1}$ and most below 200 ng $ml^{-1}$ (Wheatley, 1979). A WHO expert group (1980), on examining the reports from these studies, raised the possibility that this might be the first example of an endemic disease due to exposure to methylmercury in freshwater fish. However, another epidemiological and clinical study of the same population of Cree Indians failed to find any effects associated with methylmercury (Kaufman, personal communication to EPA), which would indicate that the background frequency of mental retardation would be increased by less than 50 percent.

Estimates of increased rates risks due to acid precipitation would depend upon a number of assumptions, including whether increases in freshwater acidity would elevate levels of methylmercury in freshwater fish and by how much, the effect of acidity on the supply of freshwater fish, as well as actions taken by local state and Federal agencies to limit fishing and sales of fish if methylmercury levels increase. Nevertheless, information on methylmercury is now reaching the point where rough estimates can be made of health risks in this country for consumption of methylmercury from freshwater fish, and information may be forthcoming on the impact of acidity on methylmercury levels in fish. At least the direction of future research is now more clear — to obtain more quantitative information on human dose-response relationship and to further test hypotheses on cause-effect relationship between pH and methylmercury levels in freshwater fish.

## 4.3 LEAD

### 4.3.1 Environmental Exposure and Uptake by Humans

4.3.1.1 Concentrations in Water — The U.S. national interim primary drinking water standard for lead is 50 μg $\ell^{-1}$. The United States Environmental Protection Agency (U.S. EPA, 1979a) summarized data in two surveys on lead in drinking water. The median lead concentration in municipal drinking water supplies is about 10 μg $\ell^{-1}$. In certain areas, such as Metropolitan Boston, drinking water may contain lead in excess of the 50 μg $\ell^{-1}$ standard. This is believed to be due to very soft water (low pH) and the presence of lead piping in the domestic water distribution system (The Nutrition Foundation Expert Advisory Committee, 1982). Lead piping is no longer used for new potable water systems in the United States (U.S. EPA, 1979a).

A recent national survey of Canadian drinking water supplies involving 71 municipalities representing 55 percent of the population, indicated a medial level of lead $\leq 1\ \mu g\ \ell^{-1}$ and values ranged from $< 1\ \mu g\ \ell^{-1}$ to $7\ \mu g\ \ell^{-1}$.

In areas where the home water supply is stored in lead lined tanks and where it is conveyed to the household taps by lead pipes, the lead concentration may reach several hundred micrograms per liter and even exceed $1000\ \mu g\ \ell^{-1}$ (Beattie et al., 1972). The concentration of lead in water conveyed through lead pipes is affected by several factors. The longer the water is held in the pipes, the higher the lead concentrations (Wong and Berrang, 1976). The so-called "first flush" sample generally has lead concentrations about three times higher than free-running tap water (Nutrition Foundation Expert Advisory Committee, 1982). The lower the pH of the water and the lower the concentration of dissolved salts, the greater the solubility of lead in water.

Leaching of lead from plastic pipes has also been reported (Heusgem and DeGraeve, 1973). The source of lead was probably lead stearate, which is used as a stabilizer in the manufacture of polyvinyl plastics.

4.3.1.2 Concentrations in Aquatic Biota -- Lead has been reported to bind to a wide range of organic fractions in river water (Ramamoorthy and Kushner, 1975). As water pH decreases the fraction of heavy metals bound to organic components also decreases and the concentration of free inorganic metal species increases. This should increase lead levels in aquatic biota, possibly affecting human dietary intake (see Sections 2.3 and 3.3).

4.3.1.3 Human Uptake and Risk Assessment — Mahaffey (1977) estimated that the daily intake of drinking water ranged from 300 ml for children to as much as 2000 ml for adults. An expert group of the National Academy of Sciences (NAS, 1980) stated a value of 1630 ml $day^{-1}$ for water intake of adults (not including amounts used to prepare foods and beverages) and a range of 100 ml to 3000 ml for children.

A study in Canada by Armstrong and McCullough quoted by the Nutrition Foundation's Expert Advisory Committee (1982) indicated that the total daily intake including water used as a food ingredient was 760 ml averaged for 0 to 6 years, and 1140 ml for the 6- to 18-year-old group. The highest average daily intake was 1570 ml for the 55 older age group. However, up to 3000 ml total water per day was consumed by some children in the 0- to 6-year-old age group and up to 4300 ml total water was consumed by certain individuals in each of the remaining age groups.

Using the NAS reported range of 100 to 3000 ml daily water intake for children and a U.S. median level of $10\ \mu g\ \ell^{-1}$ of lead, the range of intake for children would be 1 to 30 μg and for adults for 16 μg, assuming a water intake of 1600 ml $day^{-1}$ (Table 4-4). If average lead concentrations reached the interim drinking water standard of $50\ \mu g\ \ell^{-1}$, these intake values would be five times greater.

Lead levels in blood of children in the United States vary widely (Mahaffey et al., 1982a). A criterion of 30 μg Pb 100 $ml^{-1}$ whole blood has been used to indicate elevated blood lead (Center for Disease Control, 1978). If this concentration of blood lead is accompanied by an erythrocyte protoporphyrin

TABLE 4-4. DAILY INTAKE OF LEAD FROM DRINKING WATER

| Age Group | Daily Water Intake[a]<br>ml | Daily Lead Intake[b]<br>µg Pb |
|---|---|---|
| Children | 100 - 3000 | 1 - 3 |
| Adults | 1630 | 16 |

[a] National Academy of Science (1980).

[b] Assumes U.S. median concentration of lead in drinking water to be 10 µg Pb $\ell^{-1}$.

concentration of 50 to 250 μg 100 $ml^{-1}$ of whole blood, the child is thought to have undue lead absorption. Community based lead poisoning prevention programs report that approximately 75 percent of children with blood lead levels of greater than or equal to 30 μg 100 $ml^{-1}$ also have erythrocyte protoporphyrin values of greater than or equal to 50 μg 100 $ml^{-1}$ (Mahaffey et al., 1982a).

A survey of blood lead levels in children in the years 1976 to 1980 in the United States indicated that substantial numbers of children have blood leads greater than or equal to 30 μg 100 $ml^{-1}$ (Table 4-5). The prevalence of elevated blood lead values is highest in children of low income families (approximately 11 percent of children in families having an annual income less than 6,000 dollars) and in children living in large cities (7.2 percent of children living in cities of population more than one million). However, elevated blood lead is widely distributed in the general population, including children in families earning more than $15,000 annual income (1.2 percent) and in children living in rural areas (2.1 percent).

Reduced pH increases the corrosivity of water and can mobilize metals such as lead, resulting in increased concentrations in drinking water. Lead piping in home plumbing is rare and no longer used in this country except in certain parts of New England. However, lead can be mobilized from other types of piping where it is used as a solder (copper piping) or in stabilizers (certain types of plastic pipes). Homes using roof-catchment cisterns for collecting drinking water seem especially vulnerable to corrosive rain water.

In a study of 40 roof-catchment cistern systems in western Pennsylvania, Young and Sharpe (1984) report that lead in atmospheric deposition accumulates in the sediments that collect at the bottoms of cisterns and that this particulate lead could appear in the drinking water of cistern users when conditions allowing the suspension of this material in cistern water are present. They did not report on the frequency of such conditions, but they did point out that in the systems they studied there were no safeguards to prevent the ingestion of lead contaminated cistern sediments. However, cistern systems with gross particulate filters for incoming catchment runoff had much lower lead concentrations in sediments.

Young and Sharpe measured the concentrations of lead in tapwater that had stood in the cistern plumbing system overnight. In nine of the 40 systems studied (22 percent) average lead concentrations exceeded the drinking water standard of 50 μg Pb $\ell^{-1}$ (U.S. EPA, 1979b).

Taylor et al., (1984) found that lead concentrations in 7 of 129 household water samples from both surface and ground water sources exceeded the federal drinking water standard of 50 μg Pb $\ell^{-1}$ (U.S. EPA, 1979b). Violation of the drinking water standard occurred primarily in samples that had remained overnight in household plumbing.

From the point of view of human health risks, any increases of lead concentrations in drinking water should be viewed as an additional burden of lead. This is especially important with children, of whom substantial numbers already have elevated blood levels. At the median concentration of 10 μg Pb $\ell^{-1}$ drinking water already makes an appreciable contribution to blood lead levels (approximately 30 percent of the total from all other sources of lead). Thus

TABLE 4-5. BLOOD LEAD LEVELS IN CHILDREN 6 MONTHS THROUGH 5 YEARS BY ANNUAL FAMILY INCOME AND DEGREE OF URBANIZATION OF PLACE OF RESIDENCE IN THE UNITED STATES FROM 1976 TO 1980[a].

| Demographic variable | Estimated population (thousands) | No. of persons examined | Blood[b] lead μg 100 ml$^{-1}$ | Blood lead levels < 30 μg 100 ml$^{-1}$ % persons examined |
|---|---|---|---|---|
| Annual Family Income[c] | | | | |
| < $6000 | 2465 | 448 | 20 ± 0.6 | 10.9 ± 1.4 |
| $1000 - 14,999 | 7534 | 1083 | 16 ± 0.5 | 4.2 ± 0.7 |
| > 15,000 | 6428 | 774 | 14 ± 0.4 | 1.2 ± 0.4 |
| Degree of Urbanization | | | | |
| urban > $10^6$ persons | 4344 | 544 | 18 ± 0.5 | 7.2 ± 0.7 |
| urban < $10^6$ persons | 6891 | 944 | 16 ± 0.7 | 3.5 ± 0.6 |
| Rural | 5627 | 884 | 14 ± 0.6 | 2.1 ± 0.9 |

[a] Adapted from Mahaffey et al. (1982a).

[b] Mean ± S.E.M.

[c] All values shown for this variable reflect the exclusion (from analysis and tests of significance) of children in households that declined to report their income.

the drinking water standard of 50 μg Pb $\ell^{-1}$ will not provide sufficient protection to children already having elevated blood lead from other sources of exposure.

Quantitative data are lacking on the contribution of acidic deposition to lead in drinking water. Roof-catchment cistern systems believed to be widely used in rural areas of Ohio and Western Pennsylvania are likely to be affected by acidic deposition. Thus, it is of great importance to ascertain the extent to which cistern systems are used in areas of the USA subject to acidic deposition and to check the extent to which changed corrosivity of this water affects lead levels in tap water.

### 4.3.2 Effects on Human Health

4.3.2.1 Metabolism in Humans — The uptake, distribution, and excretion of lead have recently been reviewed in detail (U.S. EPA, 1980b). Approximately 8 percent of dietary lead is absorbed in the gastrointestinal tract in adults. Children absorb about 50 percent of ingested lead. Lead may be absorbed with greater efficiency from water and other beverages than from food.

Lead is distributed to all tissues in the body and to all compartments within cells. Most of the lead in blood is associated with the red blood cells. The skeleton is the main site of lead storage, with about 95 percent of the total lead in the body in the skeleton of adults. Lead readily crosses the placenta. It also crosses the blood-brain barrier but more readily in children than in adults.

Lead is excreted in urine and feces, with the human urinary route probably being more important. The half-time of lead retention in soft tissues is about six weeks following exposure of a few months. The half-time may be longer following years of occupational exposures to lead. Lead is accumulated in the skeleton throughout most of the human life-span, and the half-time in skeletal tissue is very long.

Lead concentration in whole blood is the most commonly used indicator for assessing the burden of lead in soft tissues. The relative contributions of airborne lead, lead in food, and other sources of lead are usually assessed in terms of their contributions to the blood-level concentration.

A positive correlation exists between the concentration of lead in domestic water supply and the concentration of lead in blood. The U.S. EPA, based on a study by Moore et al., (1977), has estimated blood concentrations associated with various levels of lead in free-running tap water (Table 4-6).

If the U.S. EPA estimate is valid, the impact of lead concentrations in running tapwater is greatest in the lower range of lead in water. According to Table 4-6, the median lead level in U.S. drinking water (10 μg $\ell^{-1}$) would contribute approximately 7.4 μg 100 $ml^{-1}$. Assuming the median blood level in the absence of the water contribution to be 11 μg 100 $ml^{-1}$, the lead present in tapwater at the current interim primary drinking water standard would contribute about 10 μg 100 $ml^{-1}$ to blood lead concentration, i.e., about equal to the lead contribution from all other sources. However, blood levels in the United States

TABLE 4-6. THE ESTIMATED RELATIONSHIP BETWEEN LEAD CONCENTRATIONS IN RUNNING TAP WATER AND HUMAN BLOOD LEAD LEVELS (MOORE ET AL. 1977 IN U.S. EPA 1980b).

| Lead in running tap water (μg ℓ$^{-1}$) | Total lead in blood (PbB) (μg 100 mℓ$^{-1}$) | Lead in Blood due to water (μg 100 mℓ$^{-1}$) |
|---|---|---|
| 0 | 11 | 0 |
| 1 | 14.4 | 3.4 |
| 5 | 16.7 | 5.8 |
| 10 | 18.4 | 7.4 |
| 25 | 21.0 | 10.0 |
| 50 | 23.6 | 12.6 |
| 100 | 26.8 | 15.8 |

are affected by a number of factors such as age, sex, and urban versus non-urban locations. Urinary excretion of lead may be used on a group basis to indicate the soft tissue burden. Lead in hair represents external contamination of the hair sample.

4.3.2.2 Effects and Dose-Response in Humans -- Lead damages a variety of human organs and tissues. Damage to the human hemopoietic system is usually the first observable effect of lead (Figure 4-2). The inhibition of enzymes involved in synthesizing hemoglobin results in the accumulation of precursor substances: δ-aminolevulinic acid (δ-ALA) in plasma and urine, and free erythrocyte protoporphyrin (FEP) on the red blood cells. Measuring FEP has become a routine method for checking the earliest effects of lead.

During recent years, measurement of FEP has come into wide use as the most practical screening tool both in epidemiologic studies and in monitoring populations at high risk for lead toxicity. Figure 4-3 shows the curvilinear relationship between FEP and lead concentration in blood. The curvilinear shape is typical of the relationship between blood lead and other intermediate metabolites of porphyrin synthesis, such as δ-aminolevulinic acid and urinary coproporphyrin. Thus it is difficult to assign a specific blood level of lead at which FEP or other metabolites attain abnormal values. At first, levels of FEP increase slowly with blood lead, but as blood lead rises about 40 to 50 μg 100 $ml^{-1}$ the rate of increment of FEP rises rapidly. Roels et al., (1978) defined abnormal blood FEP levels as those in excess of the upper 95 percent confidence limit of the controls and published a dose-response relationship relating blood lead levels to the frequency of individuals having FEP values equal to or in excess of their defined abnormal value (Figure 4-4). Children and adult females tend to show a greater response than adult males. This analysis indicates that most of a population having blood leads in the range of 30 to 40 μg 100 $ml^{-1}$ will have abnormally high FEP values.

Excessive doses of lead cause anemia and damage to both the peripheral and central human nervous systems (Table 4-7). The central nervous system in children appears to be more sensitive to damage from lead than the mature central nervous system. A growing body of knowledge suggests that lower blood levels of lead than those previously recognized are associated with altered neuropsychological function and intelligence deficiencies. For example, reduced general intelligence quotients, reduced auditory and speech processing, and attention deficiences have been reported in children with higher dentine lead than those with lower dentine lead (Needleman et al., 1979). In a study of 166 children whose blood lead levels ranged from 7 to 33 μg 100 $ml^{-1}$, Yule et al., (1981) reported decreases in attainment scores on tests of reading, spelling, and intelligence as blood lead levels increased. Some, but not all, of this variability was removed after influence of the social class of the subject's family was considered.

Piomelli (1980) has reported that heme synthesis is impaired in children with blood lead levels less than 30 μg 100 $ml^{-1}$ consistent with findings of Roels et al., (1978) reported in Figure 4-4. Several other metabolic changes associated with low level lead exposure of children have been identified. Plasma levels of the vitamin D metabolite, 1,25 dihydroxy vitamin D, which is active in stimulating the gastrointestinal absorption of calcium and phosphorus, decreased as the blood level increased (Rosen et al., 1980). Plasma

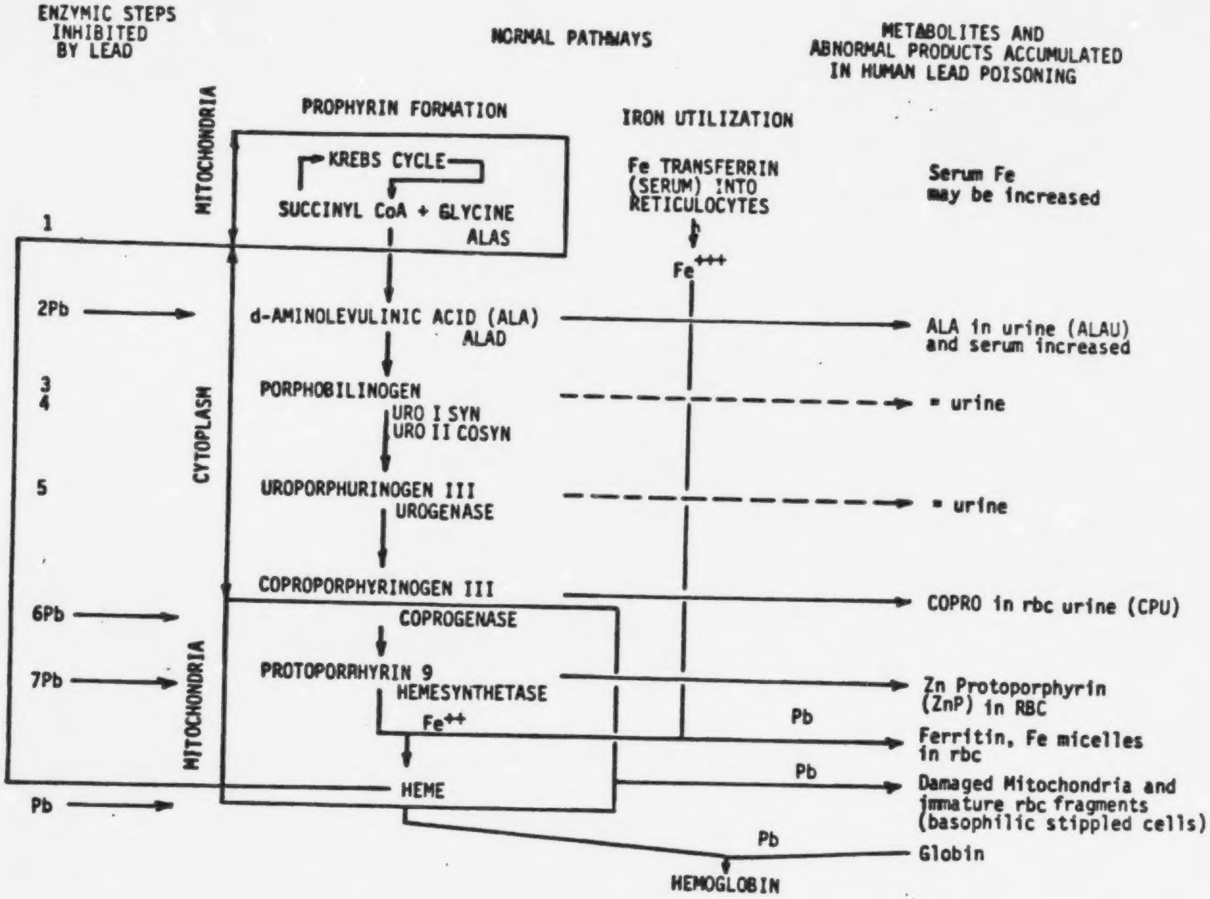
ENZYMIC STEPS INHIBITED BY LEAD
NORMAL PATHWAYS
METABOLITES AND ABNORMAL PRODUCTS ACCUMULATED IN HUMAN LEAD POISONING
PROPHYRIN FORMATION
IRON UTILIZATION
MITOCHONDRIA
KREBS CYCLE
SUCCINYL CoA + GLYCINE
ALAS
Fe TRANSFERRIN (SERUM) INTO RETICULOCYTES
Fe+++
Serum Fe may be increased
1
2Pb
d-AMINOLEVULINIC ACID (ALA)
ALAD
ALA in urine (ALAU) and serum increased
3
4
CYTOPLASM
PORPHOBILINOGEN
URO I SYN
URO II COSYN
= urine
5
UROPORPHURINOGEN III
UROGENASE
= urine
COPROPORPHYRINOGEN III
COPRO in rbc urine (CPU)
6Pb
COPROGENASE
7Pb
MITOCHONDRIA
PROTOPORPHYRIN 9
HEMESYNTHETASE
Fe++
Zn Protoporphyrin (ZnP) in RBC
Pb
Ferritin, Fe micelles in rbc
Pb
HEME
Damaged Mitochondria and immature rbc fragments (basophilic stippled cells)
Pb
Pb
Globin
HEMOGLOBIN

Figure 4-2. The initial and final steps associated with disturbances in the biosynthesis of hemoglobin due to lead are mediated by intramitochondrial enzymes and the intermediate steps by cytoplasmic enzymes. The enzymes most sensitive to lead (Steps 2 and 7) are the SH-dependent enzymes, δ-amino-levulinate dehydrase (ALAD) and heme synthetase. Accumulation of the substrates of these enzymes (ALA and FEP) is characteristic of human lead poisoning as is increased urinary coproporhyrin excretion. Although zinc protoporphyrin (ZnP) accumulates in erythrocytes in lead poisoning (and iron deficiency), it is usually measured as "free" erythrocyte protoporphyrin (FEP). Lead reduces the bioavailability of iron for heme formation. A compensatory increase in the activity of the first enzyme in the pathway, δ-amino-levulinic acid synthetase (ALAS), occurs in response to reduced heme formation. Other compensatory responses include erythroid hyperplasia, reticulocytosis and microcytosis. Non-random shortening of erythrocyte life span has been demonstrated in lead workers. Amicrocytic, hypochromic anemia results including some morphological features noted above. Adapted from Chisolm (1978).

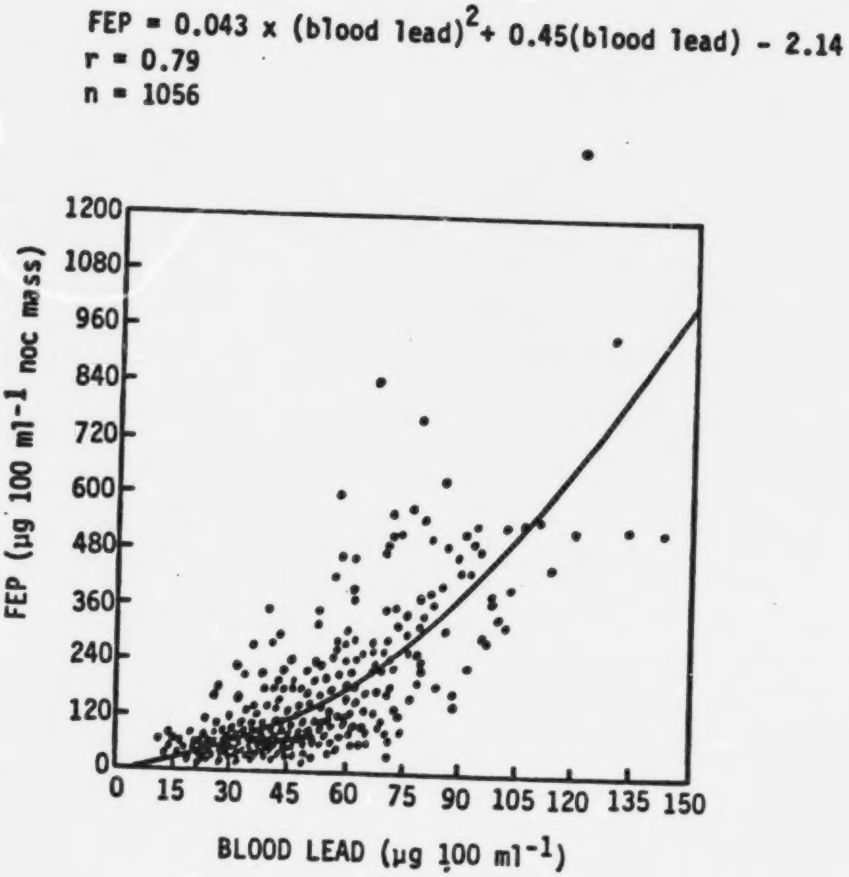

Figure 4-3. Free erythrocyte protoporphyrin (FEP) vs blood level. Shoshone County, Idaho, August 1974. Adapted from Landrigan et al. (1976).

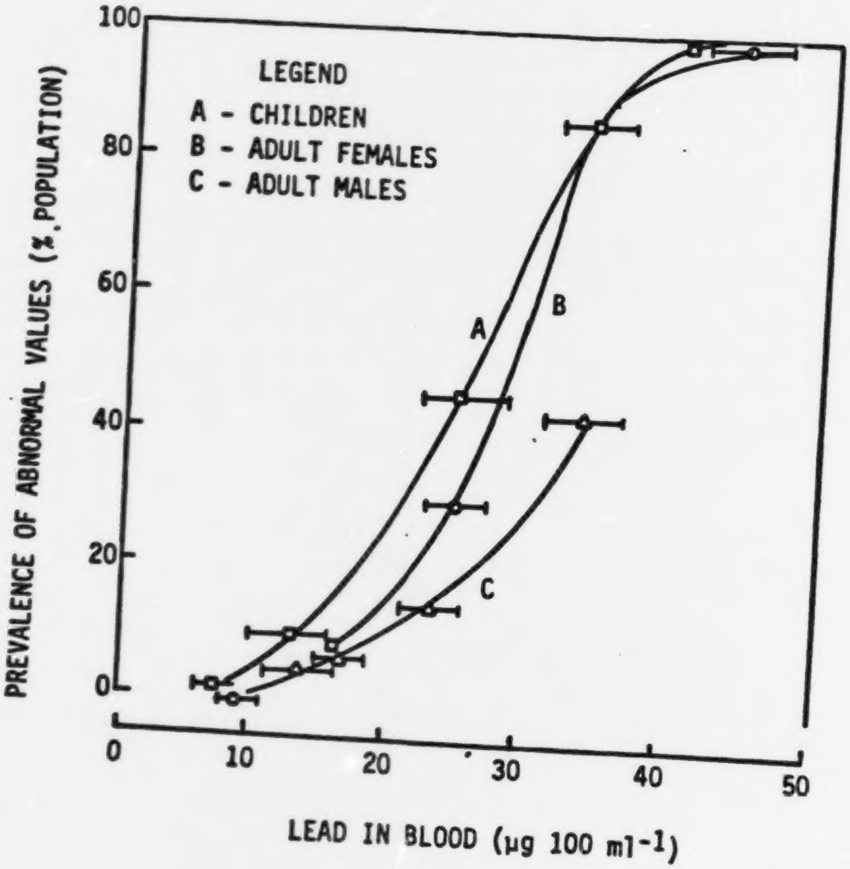

Figure 4-4. The relationship between the percentage of abnormally high free erythrocyte protoporphyrin and average blood lead levels in A, children; B, adult females; and C, adult males. Abnormal values were defined as FEP values in excess of 82, 83, and 63 µg 100 ml$^{-1}$ for children, women, and men, respectively. Adapted from Roels et al. (1978).

TABLE 4-7. NO DETECTED EFFECT LEVELS IN RELATION TO PbB (ADAPTED FROM WHO 1977)

| No-detected effect level (μg 100 $ml^{-1}$) | Effect | Population |
|---|---|---|
| < 10 | Erythrocyte ALAD inhibition | Adults, children |
| 20-25 | FEP | Children |
| 20-30 | FEP | Adults, female |
| 25-35 | FEP | Adults, male |
| 30-40 | Erythrocyte ATPase inhibition | General |
| 40 | ALA excretion in urine | Adults, children |
| 40 | CP excretion in urine | Adults |
| 40 | Anemia[a] | Children |
| 40-50 | Peripheral neuropathy | Adults |
| 50 | Anemia[a] | Adults |
| 50-60 | Minimal brain dysfunction | Children |
| 60-70 | Minimal brain dysfunction | Adults |
| 60-70 | Encephalopathy | Children |
| > 80 | Encephalopathy | Adults |

[a] The term anemia here is used to denote earliest statistically demonstrable decrease in blood hemoglobin. In adult workers a decrease in blood hemoglobin within the normal range has been reported during the first 100 days of employment. Other studies of workers indicate that frank anemia is not statistically demonstrable until PbB > 100 μg, as cited elsewhere in the WHO report. An increased frequency of early anemia has been reported at PbB > 40 μg of groups of children in whom concurrent iron deficiency anemia was ruled out but is highly likely.

levels of the vitamin D metabolite exhibited a strong negative correlation with blood lead concentrations in the range of 12 to 120 µg 100 $ml^{-1}$, with no difference in slope of the regression line from blood lead levels above or below 30 µg 100 $ml^{-1}$ (Mahaffey et al., 1982b).

Lead produces both acute and chronic effects on kidney function (Nutritional Foundation's Expert Advisory Committee, 1982). The acute effects manifested as dysfunction of the proximal tubular cell such as amino aciduria, glycoseria, and hyperphosphoturia, usually do not occur until blood levels exceed 70 µg 100 $ml^{-1}$. Chronic lead nephropathy is not usually recognized in humans until it has reached an irreversible stage. The disease is characterized by the slow development of contracted kidneys with pronounced arterioscelerotic changes, fibrosis, glomerular atrophy and hyaline degeneration of blood levels. These changes portend progressive disease sometimes resulting in acute renal failure. The duration of excessive exposure to lead is believed to play an important role in the development of the disease. Although information on blood levels is inadequate, it is unlikely that blood levels in the general child and adult populations, even in the upper 2 to 5 percentile of the "normal" U.S. range are sufficient to produce chronic renal effects.

Studies in the 19th and early 20th centuries indicated that occupational exposures to lead (presumably higher than current exposures) caused increased frequency of abortions and stillbirths (Oliver 1911). Indeed, since the publication of Oliver's findings, women were largely excluded from occupational exposures to lead until very recently.

Lancranjan et al., (1975) have reported reduction in sperm counts and abnormal sperm morphology in occupationally exposed men. The functional significance on fertility is not known.

Prenatal exposure to lead may be associated with mental retardation in children (Moore, 1980). The human data are consistent with experimental findings on animals with modestly elevated blood levels, approximately 40 µg Pb 100 $ml^{-1}$, during prenatal and early postnatal life may be associated with subtle and long lasting adverse consequences to the offspring.

Lead has been shown to be a carcinogen in animal tests, but epidemiological studies have failed to reveal an association between lead exposure and human cancer. Measurement of precursor metabolite of heme synthesis such as FEP or δ-ALA provide the earliest warning of the effects of lead. Effect on heme synthesis will protect against the more serious clinical effects of lead, such as anemia and encephalopathy.

## 4.4 ALUMINUM

### 4.4.1 Environmental Exposure and Uptake by Humans

4.4.1.1 Concentrations in Water -- Aluminum is a significant element of the earth's crust and is abundant in clay soils. While the geochemical behavior of aluminum is not precisely known, many investigators (e.g. Hutchinson et al., 1978; Hultberg and Johansson, 1981; Driscoll et al., 1980) have observed an increase in aluminum concentrations in water with decreasing solution pH. This phenomenon is consistent with theoretical and experimental solubility of aluminum minerals (see Section 2.4).

There are no synoptic data on aluminum concentration in ground and surface waters in acid sensitive regions across the United States. Initial surveys on aluminum in raw and finished drinking waters have been prepared by Fuhs et al., (1980), Taylor et al., (1984) and Miller et al., (1984). The Fuhs study was limited to several samples collected in upstate New York. The highest aluminum concentrations found in rural drinking water was 1.4 mg $\ell^{-1}$. Taylor et al. sampled 119 surface and 14 groundwater supplies in the Northeast, both raw and finished water types. This study found that the median level of aluminum in water was < 0.1 mg Al $\ell^{-1}$ with a range of < 0.1 - 0.7 mg Al $\ell^{-1}$ from 261 samples. Miller et al. collected raw and finished water samples from 186 water utilities across the United States. The survey indicated that aluminum was more likely to be found in surface waters than in groundwaters and that there was a 40 - 50 percent chance that alum coagulation would increase the aluminum concentrations in finished waters above the original concentration in the raw waters.

4.4.1.2 Concentrations in Aquatic Biota — The toxic properties of aluminum are self-limiting with regard to bioaccumulation; when the aluminum levels in water reach toxic levels, the ensuing mortality of fish stops further accumulation in aquatic food chains (see Sections 2.4 and 3.3).

4.4.1.3 Human Uptake and Risk Assessment — Acute or chronic disease in man has not been related to normal dietary intake of aluminum from food or drinking water. However, a potential risk may exist under the special circumstances of patients with compromised kidney function who undergo regular therapeutic dialysis. The average daily intake of aluminum via food and water in the U.S. is approximately 5 mg, but considerable variation may occur (Greger and Baier, 1983a). Thus drinking water containing in excess of 1 mg Al $\ell^{-1}$ could make an important contribution to an individual's total intake of aluminum. The widespread use of aluminum-containing antacids leads to a substantial amount of the population ingesting several hundred milligrams, sometimes gram amounts, per day for long periods. Driscoll et al., (1980) have reported levels of aluminum in natural bodies of freshwater in the Adirondack Region of New York State to attain values as high as 800 µg Al $\ell^{-1}$ under the influence of acidic deposition. A concentration of 50 µg $\ell^{-1}$ of aluminum in dialysis water is claimed to be dangerous (Registration Committee, European Dialysis and Transplant Association, 1980). The American National Standards Institute (1982) has established a limit of 10 µg $\ell^{-1}$ for aluminum content of dialysates.

Of the various species of aluminum known to exist in bodies of natural water, data are available only on aluminum hydroxide, which is absorbed across the human gastrointestinal tract. In areas of the country where drinking water is fluoridated or where elevated fluoride concentrations occur naturally, it is likely that aluminum fluoride complexes will be present in tap water in substantial amounts. Unfortunately, we know nothing of the gastrointestinal absorption of aluminum fluoride or about its potential toxicity in humans.

No systematic attempt has yet been made to develop quantitative estimates of populations, exposure profiles and risks. Rural populations deriving their drinking water from private shallow wells are likely to be at increased risk.

### 4.4.2 Effects on Human Health

4.4.2.1 Metabolism in Humans — Data on absorption, distribution, and excretion of aluminum compounds in man have been reviewed recently (Norseth, 1979). Aluminum is absorbed in the gastrointestinal tract. The fraction of dietary intake absorbed into the blood stream is believed to be small, but precise figures are not available. When aluminum was given as the hydroxide salt to uremic patients, approximately 15 percent of the dose was absorbed, with considerable differences among individuals (Clarkson et al., 1972). No information is available on the absorption of other forms of aluminum or on absorption in people with normal kidney function. Aluminum is distributed to all tissues in the body and has been reported in fetal tissues. When aluminum in food was given to rats, increased levels were reported in blood, brain, liver, and testes (Ondreicka et al., 1966).

Little information is available on the relative importance of urine and fecal pathways of aluminum excretion. Renal clearance of aluminum may be as high as 10 percent of the glomerular filtration rate (creatinine clearance) as indicated in patients with compromised renal function. These data would suggest a high urinary rate of excretion in normal subjects and a correspondingly short biological half-time (on the order of days or hours). Animal experiments indicate that biliary excretion of aluminum contributes to fecal excretion of the metal.

Aluminum is found in both cow and human milk. Normal levels of aluminum in human blood and other biological fluids exhibited a very wide range of values, apparently affected by differences in the laboratories making the analyses. Chance contamination by the ubiquitous metal causes important problems in determining reliable values for the low levels in human plasma.

4.4.2.2 Effects and Dose-Response in Humans — Aluminum has long been thought to be innocuous, consequently in the U.S. there are no established food or drinking water standards for this pollutant. Because of recent studies associating aluminum in water used for dialysis with senile dementia and dialysis encephalopathy, and because aluminum continues to be implicated in central nervous system-degenerative diseases such as Alzheimer's disease, the U.S. EPA has proposed establishing National Primary Drinking Water Standards for this pollutant (U.S. EPA, 1983).

Aluminum interacts with phosphate and fluoride in the gut. At intakes around 100 mg Al day$^{-1}$ phosphate absorption may be decreased by a few percent, but the regulation of phosphate by the kidneys easily compensates for that. Daily intakes of several grams of aluminum for long periods may cause phosphate depletion and has caused osteomalacia in women.

Systematic effects of aluminum absorbed from the gut have not been found in persons with good renal functions. In persons with chronic renal failure the decrease in renal elimination, possibly also increased absorption, has caused accumulation in bone and brain (Alfrey et al., 1976). In uremic patients given gram amounts of aluminum hydroxide to prevent absorption of phosphorus, this accumulation has caused osteomalacia and encephalopathy, especially in children (Alfrey, 1984). Haemodialysis with an aluminum concentration above 50 µg ℓ$^{-1}$ in the dialysate has caused several cases of lethal encephalopathy. The dialysis encephalopathy is now prevented by not allowing the water used for the dialysate to contain more than 10 µg ℓ$^{-1}$.

Aluminum has also been suspected of causing Alzheimer's disease (primary degenerative dementia), a disease of the elderly which is a common cause of death in the U.S. (Crapper et al., 1973). The most recent data indicate that patients with this disease do not have higher concentrations of aluminum in the brain than age-matched controls (Shore and Wyatt, 1983).

High intraneuronal concentrations of aluminum have been reported in persons who suffered degenerative brain disease (amyotrophic lateral sclerosis and Parkinsonism-dementia) in Guam (Perl et al., 1982). High concentrations of aluminum and low concentrations of calcium and magnesium were stated to occur in drinking water of that area. However, the concentrations of aluminum were not reported. Because of the lack of quantitative information concerning exposure the relationship between high aluminum exposure and the occurrence of degenerative brain disease cannot be regarded as established.

## 4.5 CADMIUM

### 4.5.1 Environmental Exposure and Uptake by Humans

4.5.1.1 Concentrations in Water -- Limited research to date has not documented significant quantities of cadmium in tap water for water supplies (Young and Sharpe, 1984; Taylor et al., 1984; Meranger et al., 1983). Intake via drinking water is generally less than 2 µg day$^{-1}$ (Sharrett et al., 1982). Influx of cadmium through the combination of direct input of acidic deposition and as corrosion products resulting from the contact of acidic water with household plumbing is most likely in roof-catchment cistern systems.

Young and Sharpe (1984) also report accumulations of cadmium in cistern sediments, although less frequently than lead. The cadmium concentrations in atmospheric deposition shown by the Young and Sharpe study are generally very low, indicating that some other source such as corrosion of galvanized gutters and downspouts might be present.

4.5.1.2 Concentrations in Aquatic Biota -- The solubility of cadmium and the concentration of free inorganic metal species in solution both increases as water pH decreases. This should increase cadmium levels in aquatic biota, possibly increasing human dietary intake (see Sections 2.5 and 3.3).

4.5.1.3 Human Uptake and Risk Assessment -- The average dietary intake of cadmium in the U.S. is around 20 µg, with large variations (Kowal et al., 1979; Kjellstrom, 1979; Spencer et al., 1979; Ryan et al., 1982). The cadmium levels in ambient air are generally less than 10 mg cubic meter$^{-1}$, but tobacco contains 1-2 µg g$^{-1}$ and smoking a pack of cigarettes a day may cause an absorption of the same magnitude as that from food since cadmium in the inhaled smoke may be absorbed up to 50 percent (Piscator, 1982).

### 4.5.2 Effects on Human Health

4.5.2.1 Metabolism in Humans — Normally, only a few percent of the cadmium in food is absorbed from the gastrointestinal tract, but iron deficiency (Flanagan et al., 1978) may cause considerably higher absorption, up to 20 percent. Also cadmium deficiency may increase the absorption. Absorbed cadmium is mainly deposited in the liver where it is bound to a low molecular weight protein, metallothionein, which also can bind zinc, copper and mercury.

From the liver cadmium will be slowly released with a long biological halftime, 5 - 10 years, and transported to the kidney, where it also will be stored with similar halftime. Therefore the biological half-time in the whole body may be around 20 years. If cadmium has been ingested bound to metallothionein, the uptake in the kidney will be faster.

In the kidney the highest cadmium concentrations are in the renal cortex. The average concentration in Americans is around 25 mg kg$^{-1}$ wet weight (Piscator, 1982; Kjellstrom, 1979) at age 50. In nonsmokers the concentration in the cortex is around 15 mg Cd kg$^{-1}$ wet weight and around 30 mg Cd kg$^{-1}$ wet weight in smokers (Kjellstrom, 1979). The distribution of renal cortex concentrations is log normal and in nonsmokers the upper limit is probably around 70 mg Cd kg$^{-1}$ wet weight. A 3-fold increase in dietary intake would cause a corresponding increase in renal levels of cadmium.

4.5.2.2 Effects and Dose-Response in Humans -- The critical organ is the kidney and the critical concentration in renal cortex is around 200 mg Cd kg$^{-1}$ wet weight (Roels et al., 1983). At that level the reabsorption of proteins from the glomeral filtrate may be slightly reduced, resulting in an increased urinary excretion of especially low molecular weight proteins, e.g., β-microglobulin. This effect probably occurs when the renal capacity to synthesize metallothionein and keep cadmium in an inert form is exceeded. At higher renal concentrations also other functions may be disturbed, e.g. reabsorption of calcium and phosphorus, which may cause general disturbances in bone metabolism. In combination with nutritional deficiencies the renal damage caused by cadmium was the cause of the Itai-Itai disease, an osteomalacia, in Japan.

Cadmium has also been suspected as causing cancer of the prostate, but the evidence is very weak, and there is no reason to believe that ingested cadmium has an etiological role (Piscator, 1981).

## 4.6 OTHER TRACE CONTAMINANTS

4.6.1 Asbestos -- Asbestos fibers may enter drinking water from natural erosion of asbestos-containing rock, mine processing wastes, corrosion of asbestos-cement pipe, or disintegration of asbestos tile roof used to collect and direct rain water into drinking water cisterns. While inhaled asbestos is recognized to present carcinogenic risks to human health, the effect of asbestos ingested from drinking water is unclear. The role of asbestos in the etiology of gastro- intestinal cancer has been a subject of scientific controversy. At present there is no National Primary Drinking Water Quality Standard for asbestos.

U.S. exposure to asbestos in drinking water from distribution pipes has, however, been estimated by Milette et al., (1981a, 1981b). Available data indicate that 16.5 percent of U.S. water utilities distribute highly corrosive water, and 52 percent distribute moderately corrosive water. In this same study, Millette estimated that approximately 11 percent of the U.S. population is exposed to asbestos fiber concentrations in excess of 10 million fibers $\ell^{-1}$.

Kanarek et al. (1980) published the first study concluding that a statistically significant association ($P < 0.01$, t-test for multiple regression and correlation coefficients) exists between asbestos in drinking water and cancer. The water comes from aquifers or is stored in reservoirs which have contact with naturally occurring asbestos rock. In contrast, several other studies have not found significant associations between asbestos fiber concentrations and cancer.

No data directly associate acidic deposition with asbestos-related health effects. There are several explanations for this lack of association: diagnosis of asbestos-related cancer is difficult; acidic deposition health effects and asbestos contamination of drinking water are each recently-discovered concerns and no experimental attempt has yet been made to associate them; a time lag may be involved between exposure of material to aggressive water and corrosion onset or detection; or, indeed, no such association exists.

4.6.2 Nitrates -- The incremental increase in nitrates in drinking water supplies due to deposition of nitrogen compounds is a concern due to infant methemoglobinemia from ingestion of high levels of nitrate. However, the relative importance of deposition-related nitrates to increases in nitrates from agricultural fertilizer use and sewage disposal (septic system failure, treatment plant discharges) has not been well documented. The additional input of nitrates at the Hubbard Brook watershed due to deposition since the 1960's has been estimated at about 1 mg $\ell^{-1}$ as N (National Research Council, 1983). The current U.S. maximum contaminant level for drinking water nitrates has been set at 10 mg $\ell^{-1}$ as N.

## SECTION II: SUMMARY OF RESEARCH RECOMMENDATIONS

ATMOSPHERIC PROCESSES (recommendations apply to all trace contaminants studied: mercury, lead, cadmium and aluminum).

(1) Interpret existing trace contaminant data relative to human exposure and toxicity;

(2) Evaluate the applicability of existing emissions data for the prediction of trace contaminant input into the environment;

(3) Develop techniques for measurement of dry and wet deposition (includes both vapor phase and solid phase for mercury);

(4) Establish standardized methods to include quality control procedures and archive all measurements of atmospheric concentration, dry deposition and wet deposition for trace contaminant species;

(5) Establish or coordinate a national network of trace contaminant measurements to determine their spatial variability;

(6) Evaluate the applicability of trace contaminants as unique tracers for establishing source-receptor relationships.

BIOGEOCHEMISTRY

(1) Studies of temporal and regional spatial variability in atmospheric deposition of trace metals, with common methodologies, are not available. We recommend such a study for Cd, Hg and Pb. The frequency of collection, the type of collectors, sample preservation, metal speciation and bioavailability should be addressed. A proposal of this type of survey work was prepared by National Atmospheric Deposition Program personnel and has been under consideration by the U.S. Environmental Protection Agency and U.S. Geological Survey for two years. We strongly urge consideration of this proposal or one similar to it.

(2) Taylor et al. (1984) have studied water quality in the northeastern U.S. in public water supply systems. Their study investigated water quality from raw sources to the domestic; thus it evaluated effects of acidic deposition, water treatment, and distribution systems on the water quality. A large fraction of the population in predominantly rural states (e.g. approximately 1/3 in Maine) uses water directly from surface sources, shallow dug wells or individual drilled wells - all without chemical treatment. Hultberg and Johansson (1981) studied the water quality of private dug and drilled wells in southwestern Sweden and found significant water acidification and associated high concentrations of heavy metals and Al in the raw water. Effects due to local distribution systems were also observed. A study of this type should be conducted on a regional or state-by-state basis.

(3) Given the general relationship between mercury in fish and lakewater pH, our lack of knowledge of the extent of mercury contamination of fish stocks in remote lakes in the U.S., and our present inability to predict mercury burdens in fish in such lakes, we recommend that a

national survey of mercury content in common native fish species be undertaken, with particular reference to differences in:

- watershed geology, soil type and soil depth;
- lakewater retention time;
- acidity;
- lake trophic status;
- color (humic vs. clearwater lakes);
- mercury content in sediment and watershed soil.

The lakes should be selected as a subset of those initially sampled in the U.S. National Lake Survey, Phase I; the proposed mercury study could be incorporated into Phase II of the National Lake Survey (biological parameters).

(4) Recent experimental results (Hultberg, personal communication) indicate that biotic transformations of mercury may have an important influence on mercury bioaccumulation (e.g. chemical methylation; volatilization). Experiments should be undertaken to determine whether or not chemical methylation occurs in the natural environment (soils, sediments, surface waters) and to evaluate its relative importance as a function of such factors as pH, fulvic acid concentration, iron and manganese levels.

(5) Biogeochemical cycling of mercury is poorly understood and thus predictions about fluxes, body burdens in fish, altered pathways (due to acidification), and related questions cannot be adequately addressed. To better understand the biogeochemical cycling of mercury we recommend that the mercury flux through the ecosystem be studied at calibrated watershed sites for a small number of lakes which differ in:

- productivity;
- hydrology;
- atmospheric loading of mercury;
- acidity.

Depending on available funding the study could look at a variety of processes, including:

- relative importance of wet and dry deposition;
- chemical form of mercury in atmospheric deposition;
- mercury fluxes between terrestrial, aquatic, biological, sedimentary, and atmospheric compartments;
- transformations of mercury (reduction, methylation) within compartments;
- mechanisms of mercury uptake by fish;
- food chain interactions with mercury forms;
- methylmercury partitioning (sediment/water; soils/water; periphyton/water).
- degree of mercury adsorption/precipitation in sediments.

Intensively studied sites should include the Little Rock Lake Acidification Project in Wisconsin (U.S. EPA-funded) and an additional site (soon to be named) in the northeastern U.S. Additionally, several presently acidified lakes that are to be limed should be studied. Additional funding should be provided for these studies at the lake manipulation sites.

## BIOACCUMULATION

(1) In Scandinavia, Canada, and the United States, mercury concentrations exceeding 1.0 µg $g^{-1}$ wet weight in piscivorous fishes have been recorded, particularly in low pH lakes. However, there are no comprehensive surveys of mercury contamination of fish across different geographic regions containing susceptible softwater lakes. Development and extensive statistical analysis of a centralized data base on mercury concentrations in fish in acid-sensitive lakes that lack direct or local anthropogenic sources of mercury is needed to define the geographic extent of this problem. In addition, Phase II of the (U.S.) National Lake Survey should include sampling and analysis of fish for total mercury concentration in axial muscle tissue. A subset of the fish samples should be analyzed for methylmercury.

(2) Mechanisms enhancing availability of methylmercury in poorly buffered lakes should be elucidated. At a minimum, whole-lake experimental acidification studies in the Upper Midwest and the northeastern United States should include collection and analysis of fishes for total mercury concentration at annual intervals.

(3) To evaluate the effect of chemical neutralization on mercury contamination of fishery resources, Task Group E (Objective E-5) of the National Acid Precipitation Assessment Program should consider inclusion of mercury analysis of fishes and other biota in recently initiated and future mitigation studies.

## INDIRECT HEALTH EFFECTS

(1) Freshwater fish with the highest user consumption figures and the highest average methylmercury levels are among the most likely species to be affected by acidic deposition (see Section 4.2.1.3). More research is needed to delineate the cause-effect relationship between acidic deposition, pH, and methylmercury levels in freshwater fish.

(2) Human patterns of freshwater fish consumption need documentation so that populations at risk for methylmercury toxicity may be identified. With these data, an average methylmercury intake in freshwater fish consumers could be calculated.

(3) Prenatal life is a more sensitive stage of the life cycle for methylmercury (see Section 4.2.2.2). More information is needed on fish consumption patterns of women of child-bearing age in order to

quantitatively assess the potential impact on human health of elevated methylmercury levels in freshwater fish.

(4) Populations are at increased risk of being exposed to higher concentrations of corrosive toxicants, such as lead and possibly cadmium, where acidic surface waters are used for drinking without corrosive control (see Section 4.3.1.3). Quantification of the extent of lead and cadmium mobilization from water distribution systems and domestic plumbing into drinking water is warranted. The populations at risk and water distribution networks involved need to be identified.

(5) People receiving drinking water from roof catchment cisterns are at potential risk of increased intake of lead and possibly cadmium in areas of acidic deposition (see Section 4.3.1.3). The population at risk needs to be identified and quantification of the extent of lead and cadmium mobilization from roof catchments systems and domestic plumbing is warranted.

(6) Individual water supplies emanating from shallow groundwater in areas edaphically and geologically sensitive to acidic deposition, may contain elevated concentrations of aluminum. Quantification of the extent of aluminum contamination in these water supplies and identification of populations at risk is warranted.

(7) The human toxicity of lead has been extensively studied (see Section 4.3.2.2). However, further study is necessary concerning lead effects on certain physiological processes.

- Determine the functional significance of lead exposure on infertility in the human population.

- Determine the effect of prenatal exposure to lead on the development of the fetus.

(8) Aluminum toxicity to humans is of growing concern, particularly due to its association with certain types of dementia: dialysis dementia, senile dementia, Alzheimer's disease, and Parkinson's disease (see Section 4.4.2.2). The following studies are warranted.

- Pharmacokinetic studies are needed in appropriate animal models to establish the relationship between oral intake of aluminum and the levels of aluminum in brain tissue. These data, when obtained, should be extrapolated to man.

- Biochemical studies are needed to elucidate the mechanism whereby aluminum can produce chronic degenerative disease of the human nervous system.

- Studies to further clarify the relationship between dialysis and aluminum toxicity are required.

- Development of reliable analytical methods for measuring low levels of aluminum in human plasma are necessary.

  - Studies to determine the significance of osteomalacia in women with large daily intaks of aluminum with phosphate depletion are required.

(9) Acidification of certain water supplies with cement-asbestos pipe distribution systems may release asbestos fibers into the water (see Section 4.6.1). The following studies are necessary to assess the significance of this problem.

  - Epidemiological and toxicological studies need to be conducted to ascertain the relationship between asbestos fiber ingestion and gastro-intestinal cancer.
  - Studies to quantify the extent to which acidified water is capable of mobilizing asbestos fibers from cement-asbestos piping is required.
  - The populations at risk and water distribution networks involved with asbestos contamination need to be identified.

# SECTION III: LITERATURE CITED

## REFERENCES

Abernathy, A.R., and P.M. Cumbie. 1977. Mercury accumulation by largemouth bass (Micropterus salmoides) in recently impounded reservoirs. Bull. Environ. Contam. Toxicol. 17:595-602.

Ahmad, I., Y.K. Chau, P.T.S. Wong, A.J. Carty, and L. Taylor. 1980. Chemical alkylation of lead (II) salts to tetraalkyllead (IV) in aqueous solution. Nature 287:716-717.

Akagi, H., D.C. Mortimer, and D.R. Miller. 1979. Mercury methylation and partition in aquatic systems. Bull. Environ. Contam. Toxicol. 23:372-376.

Akielaszek, J.J., and T.A. Haines. 1981. Mercury in the muscle tissue of fish from three northern Maine lakes. Bull. Environ. Contam. Toxicol. 27:201-208.

Alfrey, A.C., G.R. LeGendre and W.D. Kaehny. 1976. The dialysis encephalopathy syndrome: Possible aluminum intoxication. New Eng. J. Med. 294:184-188.

Alfrey, A.C. 1984. Aluminum intoxication. New Eng. J. Med. 310:1113-1114.

Almer, B., W. Dickson, C. Ekstrom, and E. Hornstrom. 1978. Sulfur pollution and the aquatic ecosystem. pp. 271-311 in Sulfur in the Environment, Part II, Ecological Impacts, J.O. Nriagu, ed. New York: Wiley Interscience.

American National Standard for Hemodialysis Systems. American National Standards Institute, New York, NY, May 14, 1982.

Andren, A.W., S.E. Lindberg, and L.C. Bate. 1975. Atmospheric input and geochemical cycling of selected trace elements in Walker Branch Watershed. Oak Ridge National Lab., Environ. Sci. Div. Publ. No. 728, 68 pp.

Baker, J.P., and C.L. Schofield. 1982. Aluminum toxicity to fish in acidic waters. Water Air Soil Pollut. 18:289-309.

Bakir, F., S.F. Damluji, L. Amin-Zaki, M. Murtadha, A. Khalidi, N.Y. Al-Rawi, S. Tikriti, H.I. Dhahir, T.W. Clarkson, J.C. Smith, and R.A. Doherty. 1973. Methylmercury poisoning in Iraq. Science 181:230-241.

Barrie, L.A., R.M. Hoff, and S.M. Daggupaty. 1981. The influence of mid-latitudinal pollution sources on haze in the Canadian Arctic. Atmos. Environ. 15:1407-1419.

Beattie, A.D., M.R. Moore, W.T. Devenay, A.R. Miller, and A. Goldberg. 1972. Environmental lead pollution in an urban soft-water area. Brit. Med. J. 2:4901-4903.

Billen, G., C. Joiris, and R. Wollast. 1974. A bacterial methylmercury mineralizing activity in river sediments. Water Res. 8:219-225.

Bishop, P.L., and E. Kirsch. 1972. Biological generation of methylmercury in anaerobic pond sediment. Eng. Bull. Purdue Univ. Series 1, 14-1 pt. 2. 628-638.

Bloomfield, J.A., S.O. Quinn, R.J. Scrudato, D. Long, A. Richards, and F. Ryan. 1980. Atmospheric and watershed inputs of mercury to Cranberry Lake, St. Lawrence County, New York. Pages 175-207 In T.Y. Toribara, M.W. Miller, and P.E. Morrow, editors. Polluted rain. Plenum Press, New York, USA.

Bodaly, R.A., R.E. Hecky, and R.J.P. Fudge. 1984. Increases in fish mercury levels in lakes flooded by the Churchill River diversion, northern Manitoba. Can. J. Fish. Aquat. Sci. 41:682-691.

Bollingberg, H.J., and P. Johansen. 1979. Lead in spotted wolffish, Anarhichas minor, near a zinc-lead mine in Greenland. J. Fish. Res. Board Can. 36:1023-1028.

Brouzes, R.J.P., R.A.N. McLean, and G.H. Tomlinson. 1977. The link between pH of natural waters and the mercury content of fish. Research Report. Senneville, Quebec, Domtar Research Centre.

Buergel, P.M., and R.A. Soltero. 1983. The distribution and accumulation of aluminum in rainbow trout following a whole-lake alum treatment. J. Freshwater Ecol. 2:37-44.

Calvert, J., J.M. Galloway, J.M. Hales, G.M. Hidy, J. Jacobson, A. Lazrus, J. Miller, V. Mohnen, and M.F. Uman. 1983. Acid Deposition: Atmospheric processes in eastern North America. National Academy Press, Washington, DC. p. 375.

Center for Disease Control. 1978. Preventing lead poisoning in young children. J. Pediatr. 93:709-720.

Chisolm, J.J., Jr. 1978. Heme metabolite blood and urine in relation to lead toxicity and their determination. Clinical Chem. 20:225-265.

Church, T.M., J.M. Tramontano, J.R. Scudlark, T.D. Jickells, J.J. Tokos, Jr., and A.H. Knapp (1985). The wet deposition of trace metals to the western Atlantic Ocean at the mid-Atlantic coast and on Bermuda. Atm. Env. (In Press).

Clarkson, E.M., V.A. Luch, W.V. Hynson, R.R. Bailey, J.B. Eastwood, J.S. Woodlead, V.R. Clements, J.L.H. O'Riodan, and H.E. DeWardener. 1972. The effect of aluminum hydroxide on calcium, phosphorus and aluminum balances. Clin. Sci. 43:519-531.

Clarkson, T.W. 1983. Dynamics and toxicity in humans (mercury). Pages 24-34 In The acidic deposition phenomenon and its effects: Critical assessment review papers, Volume II--Effects sciences, Chapter 6--Indirect effects on health. U.S. Environmental Protection Agency, EPA-600/8-83-016B, Washington, DC, USA.

Cordle, F., P. Corneliusjen, C. Jelinck, B. Hacklay, R. Lehman, Jr. McLaughlin, R. Rhodes, and R. Shapiro. 1979. Human exposure to polychlorinated biphenyls and polybrominated biphenyls. Environ. Health Perspectives 24:157-173.

Crapper, D.R., S.S. Krishnan and A.J. Dalton. 1973. Brain aluminum distribution in Alzheimer's disease and experimental neurofibrillary degeneration. Science 180:511-512.

Cronan, C.S., and C.L. Schofield. 1979. Aluminum leaching response to acid precipitation: effects on high elevation watersheds in the Northeast. Science 204:304-306.

Davidson, C.I., J.M. Miller, and M.A. Pleskow. 1982. The influence of surface structure on predicted particle dry deposition to natural grass canopies. Water Air Soil Pollut. 18:25-43.

Davidson, C.I., S. Santhanam, R.C. Fortmann, and M.P. Olson. 1984a. Atmospheric transport and deposition of trace elements, $SO_4^{2-}$, and $NO_3^-$ onto the Greenland Ice Sheet. Submitted to Atmos. Environ.

Davidson, C.I. S.E. Lindberg, J.A. Schmidt, L.G. Cartwright, and L.R. Landis. 1984b. Dry deposition of $SO_4^{2-}$ onto surrogate surfaces. J. Geophys. Res. (in press).

Davidson, C.I., and J.F. Osborn. 1985a. The sizes of airborne trace metal-containing particles. *In* Toxic Metals in the Air, J.O. Nriagu and C.I. Davidson, eds. J. Wiley, New York. (in press).

Davidson, C.I., G.B. Wiersma, K.W. Brown, W.D. Goold, T.P. Mathison, and M.T. Reilly. 1985b. Airborne trace elements in Great Smoky Mountains, Olympic, and Glacier National Parks. Environ. Sci. Technol. (in press).

Davis, A.O., J.N. Galloway, and D.K. Nordstrom. 1982. Lake acidification: its effect on lead in the sediment of two Adirondack lakes. Limnol. Oceanogr. 27:163-167.

Dickson, W. 1980. Properties of acidified waters. pp. 75-83 in Ecological Impact of Acidic Precipitation, D. Drablos and A. Tollan, eds. Oslo, Norway: SNSF Project.

D'Itri, F.M., C.S. Anett, and A.W. Fast. 1971. Comparison of mercury levels in an oligotrophic and an eutrophic lake. J. Mar. Technol. Soc. 5(6):10-44.

Dillon, P.J., and R.D. Evans. 1982. Whole-lake lead burdens in sediments of lakes in southern Ontario, Canada. Hydrobiologia 91:121-130.

Driscoll, C.T., J.P. Baker, J.J. Bisogni, and C.L. Schofield. 1980. Effect of aluminum speciation on fish in dilute acidified waters. Nature 284:161-164.

Drummond, R.A., G.F. Olson, and A.R. Batterman. 1974. Cough response and uptake of mercury by brook trout, *Salvelinus fontinalis*, exposed to mercuric compounds at different hydrogen-ion concentrations. Trans. Am. Fish. Soc. 103(2):244-249.

Engel, L.W., W.G. Sunda, and B.A. Fowler. 1981. Factors affecting trace metal uptake and toxicity to estuarine organisms. I. Environmental parameters, pp. 127-144 in Biological Monitoring of Marine Pollutants, F.J. Vernberg, A. Calabrese, F.P. Thurberg, and W.B. Vernberg, eds. New York: Academic Press, Inc.

Evans, H.E., P.J. Smith, and P.J. Dillon. 1983. Anthropogenic zinc and cadmium burdens in sediments of selected southern Ontario Lakes. Can. J. Fish. Aquat. Sci. 40:570-579.

Fitzgerald, W.FG., G.A. Gill, and A.D. Hewitt. 1983. Air-c exchange of mercury. In Wong et al., editors. Trace metals in seawater. Plenum Press, New York, USA.

Fagerström, T., and A. Jernelöv. 1972. Some aspects of the quantitative ecology of mercury. Water Res. 6:1193-1202.

Fjerdingstad, E., and J.P. Nilssen. 1983. Heavy metal distribution in Norwegian acidic lakes: A preliminary record. Arch. Hydrobiol. 96(2):190-204.

Flanagan, P.R., J.S. McLellan, G. Cherian, M.J. Chamberlain, and L.S. Valberg. 1978. Increased dietary cadmium absorption in mice and human subjects with iron deficiency. Gastroenterology 74:841-846.

Fuhs, G.W., M.M. Reddy, and P.P. Parekh. 1980. Distribution of mercury and 14 other elements in remote watersheds in the Adirondack Mountains, NY. Presented at the Second Chemical Congress of the North American Continent, Las Vegas, NV, August 24-29.

Fuller, W.H., N.E. Korte, E.E. Niebla and B.A. Alesh. 1976. Contribution of the soil to the migration of certain common and trace elements. Soil Sci. 122:223-235.

Furutani, P.A., and J.W.M. Rudd. 1980. Measurement of mercury methylation in lake water and sediment samples. Appl. Environ. Microbiol. 40:770-776.

Galloway, J.N., S.J. Eisenreich and B.C. Scott. 1980. Toxic substances in atmospheric deposition: a review and assessment. National Atmospheric Deposition Program, NC-141, Univ. Virginia, Charlottesville, VA.

Galloway, J.N., J.D. Thornton, S.A. Norton, H.L. Volchok, and R.A.N. McLean. 1982. Trace metals in atmospheric deposition: A review and assessment. Atmos. Environ. 16:1677-1700.

Gardiner, J. 1974. Chemistry of cadmium in natural water. I. Study of cadmium complex formation using the cadmium specific-ion electrode. Water Res. 8:23-30.

Garland, J.A. 1978. Dry and wet removal of sulfur from the atmosphere. Atmos. Environ. 12:349-362.

Glazer, R., and D. Bohlander. 1978. Mercury levels in fish from eleven northeastern Minnesota lakes, 1977. Minnesota Department of Natural Resources, Invest. Report No. 355, St. Paul, Minnesota, USA.

Göthberg, A. 1983. Intensive fishing--A way to reduce the mercury level in fish. Ambio 12(5):259-261.

Greger, J.L., and M.J. Baier. 1983. Metabolism of aluminum by human subjects fed two levels of aluminum. Food Chem. Toxicol. 21:473-477.

Guy, R.D., and C.L. Chakrabarti. 1976. Studies of metal-organic interactions in model systems pertaining to natural waters. Can. J. Chem. 54:2600-2611.

Haines, T.A. 1981. Acidic precipitation and its consequences for aquatic ecosystems: A review. Trans. Am. Fish. Soc. 110:669-707.

Hakanson, L. 1974. Mercury in Lake Vänern--present status and prognosis (in Swedish). SNV PM 563/NLU Rapport 80, Uppsala, Sweden.

Hakanson, L. 1980. The quantitative impact of pH, bioproduction and Hg-contamination on the Hg-content of fish (pike). Environ. Pollut. (Series B) 1:285-304.

Hall, R.J., G.E. Likens, S.B. Fiance, and G.R. Hendrey. 1980. Experimental acidification of a stream in the Hubbard-Brook Experimental Forest, New Hampshire. Ecology 61:976-989.

Hanson, D.W., S.A. Norton, and J.S. Williams. 1982. Modern and paleolimnological evidence for accelerated leaching and metal accumulation in soils in New England by atmospheric deposition. Water Air Soil Pollut. 18:227-239.

Heliwell, S., G.E. Batley, T.M. Florence, and B.G. Lumsden. 1983. Speciation and toxicity of aluminum in a model fresh water. Environ. Technol. Lett. 4:141-144.

Heusgem, C., and J. DeGraeve. 1973. Importance de l'apport ailmentaire en plomb l'est de la Belgique, p. 85. *In* Proc. Int. Symp. Environ. Health Aspects of Lead. Amsterdam, 2-6 October 1972. Comm. Eur. Comm. Luxembourg.

Hodson, P.V., B.R. Blunt, and D.J. Spry. 1978a. Chronic toxicity of waterborne and dietary lead to rainbow trout (*Salmo gairdneri*) in Lake Ontario water. Water Res. 12:869-878.

Hodson, P.V., B.R. Blunt, and D.J. Spry. 1978b. pH-induced changes in blood lead of lead-exposed rainbow trout (*Salmo gairdneri*). J. Fish. Res. Board Can. 35:437-445.

Holcombe, G.W., D.A. Benoit, E.N. Leonard, and J.M. McKim. 1976. Long-term effects of lead exposure on three generations of brook trout (*Salvelinus fontinalis*). J. Fish. Res. Board Can. 33:1731-1741.

Huber, F., and U. Schmidt. 1976. Methylation of organolead and lead (II) compounds to $(CH_3)_4Pb$ by microorganisms. Nature (London) 259:157-158.

Huckabee, J.W., J.W. Elwood, and S.G. Hildebrand. 1979. Accumulation of mercury in freshwater biota. Pages 277-302 *In* J.O. Nriagu, editor. The biogeochemistry of mercury in the environment. Elsevier/North-Holland Biomedical Press, New York, USA.

Huckabee, J.W., S.A. Janzen, B.G. Blaylock, Y. Talmi, and J.J. Beauchamp. 1978. Methylated mercury in brook trout (*Salvelinus fontinalis*): Absence of an in vivo methylating process. Trans. Am. Fish. Soc. 107:848-852.

Hultberg, H. personal communication. Swedish Environmental Research Institute, Gothenburg, Sweden.

Hultberg, H. and A. Wenblad. 1980. Acid groundwater in southwestern Sweden. Pages 220-221 In Ecological Impact of Acid Precipitation. SNSF Project, Oslo, Norway.

Hultberg, H., and B. Hasselrot. 1981. Mercury in the ecosystem (in Swedish). Pages 33-35 In Project coal, health and environment. Swedish State Power Board, S-16287 Vallingby, Sweden.

Hultberg, H., and S. Johansson. 1981. Acid groundwater. Nordic Hydrol. 12:51-64.

Husain, L. Chemical elements traces pollutant transport to a rural area (in press). In Toxic Metals in Air, J.O. Nriagu and C.I. Davidson, eds. John Wiley & Sons, New York.

Husain, L., and P.J. Samson. 1979. Long-range transport of trace elements. J. Geophys. Res. 84:1237-1240.

Hutchinson, T., W. Gizyn, M. Havas, and V. Zobens. 1978. Effects of long-term lignite burns on Arctic ecosystem of the Smoking Hills, pp. 317-332. In Trace Substances in Environmental Health XII. Hemphill, ed., University of Missouri, Columbia, MO.

Jaakkola, T., H. Takahashi, R. Soininen, K. Rissanen, and J.K. Miettinen. 1972. Cadmium content of sea water, bottom sediment and fish, and its elimination rate in fish. pp. 69-75 In Radiotracer studies of chemical residues in food and agriculture. International Atomic Energy Agency, Vienna.

Jackson, T.A., G. Kipphut, R. Hesslein, and D.W. Schindler. 1980. Experimental study of trace metal chemistry in soft-water lakes at different pH levels. Can. J. Fish. Aquat. Sci. 37:387-402.

Jarvie, A.W.P., and A.P. Whitmore. 1981. Methylation of elemental elemental lead and lead (II) salts in aqueous solution. Environ. Technol. Lett. 2:197.

Jarvie, A.W.P., A.P. Whitmore, R.N. Markall, and H.R. Potter. 1983. Lead biomethylation, an elusive goal. Environ. Pollut. (Series B) 6:81-94.

Jernelöv, A. 1980. The effects of acidity on the uptake of mercury in fish. Pages 211-217 In T.Y. Toribara, M.W. Miller, and P.E. Morrow, editors. Polluted rain. Plenum Press, New York, USA.

Jernelöv, A., L. Landner, and T. Larsson. 1975. Swedish perspectives on mercury pollution. J. Water Pollut. Control Fed. 47:810-822.

Jernelöv, A., and A.L. Martin. 1980. Mercury in freshwater systems. Swedish Water and Air Pollution Research Institute, Publication B 550.

Johnson, N.M., C.T. Driscoll, J.S. Eaton, G.E. Likens, and W.B. McDowell. 1981. Acid rain, dissolved aluminum and chemical weathering at the Hubbard Brook Experimental Forest, New Hampshire. Geochim. Cosmochim. Acta 45:1421-1437.

Kahl, J.S., and S.A. Norton. 1983. Metal input and mobilization in two acid-stressed lake watersheds in Maine. Compl. Rep. A-053-ME, U.S. Dept. Int., 70 p.

Kanarek, M.S., P.M. Conforti, L.A. Jackson, R.C. Cooper, and J.C. Murchio. 1980. Asbestos in drinking water and cancer incidence in the San Francisco Bay area. Am. J. Epidemiol. 112:54-72.

Kelly, C.A., J.W.M. Rudd, R.B. Cook, and D.W. Schindler. 1982. The potential importance of bacterial processes in regulating rate of lake acidification. Limnol. Oceanogr. 27:868-882.

Kelly, T.M., J.D. Jones, and G.R. Smith. 1975. Historical changes in mercury contamination in Michigan walleyes (Stizostedion vitreum vitreum). J. Fish. Res. Board Can. 32:1745-1754.

Kelso, J.R.M., and J.M. Gunn. 1984. Responses of fish communities to acidic waters in Ontario. Pages 105-115 In G.R. Hendrey, ed. Early biotic responses to advancing lake acidification. Butterworth Publishers, Boston, Massachusetts, USA.

Kelso, J.R.M., R.J. Love, J.H. Lipsit, and R. Dermott. 1982. Chemical and biological status of headwater lakes in the Sault Ste. Marie District, Ontario. Pages 165-207 In F.M. D'Itri, ed. Acid precipitation: Effects on ecological systems. Ann Arbor Science, Ann Arbor, Michigan, USA.

Kelty, K.C., and R.G. Miller. 1981. Analysis of aluminum in water by flameless atomic absorption spectrophotometry. Proc. American Water Works Association (WQTC) Seattle, Wash.

Kjellstrom, T. 1979. Exposure and accumulation of cadmium in populations from Japan, the United States and Sweden Environ. Health Perspect. 28:169-197.

Kowal, N.E., D.E. Johnson, D.F. Kraemer, and H.R. Pahren. 1979. Normal levels of cadmium in diet, urine, blood and tissues of inhabitants of the United States. J. Toxicol. Environ. Health 5:995-1014.

Kudo, A., D.R. Miller, H. Akagi, D.C. Mortimer, A.S. De Freitas, H. Nagase, D.R. Townsend, and R.G. Warnock. 1978. The role of sediments on mercury transport (total- and methyl-) in a river system. Prog. Water Technol. 10:329-339.

Kudo, A., H. Nagase, and Y. Ose. 1982. Proportion of methyl mercury to the total amount of mercury in river waters in Canada and Japan. Water Res. 16:1011-1015.

Kumada, H., S. Kimura, and M. Yokote. 1980. Accumulation and biological effects of cadmium in rainbow trout. Bull. Jap. Soc. Sci. Fish. 46:97-103.

Kuntz, G. 1983. Personal communication, Project Engineer, Garrett Fleming Water Resources Engineers, Harrisburg, PA.

Lancranjan, J., H.I. Popescu, O. Gavanescu, I. Klepsch, and M. Serbanerscu. 1975. Reproductive ability of workmen occupationally exposed to lead. Arch. Environ. Health 30:396-401.

Landrigan, P.J., E.L. Baker, R.G. Seltmann, D.H. Cox, K.V. Bdem, W.A. Orenstein, J.A. Mather, J.A. Yankel, and I.H. Van Linder. 1976. Increased lead absorption with anemia and slow nerve conduction in children near a lead smelter. J. Pediatrics 89:904-910.

Lantzy, R.J., and F.T. Mackenzie. 1979. Atmospheric trace metals: global cycles and assessment of man's impact. Geochim. Coscmchim. Acta 43:511-525.

Lindberg, S.E. 1982. Factors influencing trace metal, sulfate and hydrogen ion concentrations in rain. Atmos. Environ. 16:1701-1709.

Lindberg, S.E. 1985. Mercury vapor in the atmosphere: Three case studies on emission, deposition, and plant uptake (in press). In Toxic Metals in the Air, J.O. Nriagu and C.I. Davidson eds. J. Wiley & Sons, New York.

Lindberg, S.E., R.C. Harriss, and R.R. Turner. 1982. Atmospheric deposition of metals to forest vegetation. Science 215: 1609-1611.

Lindqvist, O., A. Jernelöv, K. Johansson and H. Rodhe. 1984. Mercury in the Swedish environment: global and local sources. National Swedish Environment Protection Board Report, SNV PM 1816 Solna, Sweden.

Lodenius, M., A. Seppanen, and M. Herranen. 1983. Accumulation of mercury in fish and man from reservoirs in northern Finland. Water Air Soil Pollut. 19:237-246.

MacCrimmon, H.R., C.D. Wren, and B.L. Gots. 1983. Mercury uptake by lake trout, Salvelinus namaycush, relative to age, growth, and diet in Tadenac Lake with comparative data from other PreCambrian Shield lakes. Can. J. Fish. Aquat. Sci. 40:114-120.

Mahaffey, K.R. 1977. Quantities of lead producing health effects in humans: Sources and bioavailability. Environ. Health Perspect. 19:285.

Mahaffey, K.R., J.L. Annest, J. Roberts and R.S. Murphy. 1982a. National estimates of blood lead levels: United States, 1976-1980. New Eng. J. Med. 307:573-579.

Mahaffey, K.R., J.F. Rosen, R.W. Chesney, T.J. Peeler, C.M. Smith, and H.F. DeLuca. 1982b. Association between blood lead concentration and plasma 1,25-dihydroxycholecalciferol levels in children. Am. J. Clin. Nutr. 35:1327-1331.

McKim, J.M., G.F. Olson, G.W. Holcombe, and E.P. Hunt. 1976. Long-term effects of methylmercuric chloride on three generations of brook trout (Salvelinus fontinalis): Toxicity, accumulation, distribution, and elimination. J. Fish. Res. Board Can. 33:2726-2739.

McLean, R.A.N., M.O. Farkas, and D.M. Findlay. 1980. Determination of mercury in natural waters. sampling and analysis methods, pp. 151-174. In Polluted Rain. T.Y. Toribara, M.W. Miller and P.E. Morro, eds. Plenum Press, New York.

Meranger, J.C., T.R. Khan, C. Vairo, R. Jackson, and W.C. Li. 1983. Lake water acidity and the quality of pumped cottage water in selected areas of northern Ontario. Intern. J. Environ. Anal. Chem. 15(3):185-212.

Merlini, M., and G. Pozzi. 1977. Lead and freshwater fishes: Part 1--Lead accumulation and water pH. Environ. Pollut. 12:167-172.

Methyl Mercury Study Group. 1980. Methyl mercury study. McGill Univ., Montreal, Canada.

Miller, D.R., and H. Akagi. 1979. pH affects mercury distribution, not methylation. Ecotoxicol. Environ. Safety 3:36-38.

Miller, R.G., F.C. Kopfler, K.C. Kelty, J.A. Stober, and N.S. Ulmer. 1984. The occurrence of aluminum in drinking water. J. Am. Water Works Assoc. Volume:84-91.

Millette, J.R., M.F. Pansing, and R.L. Boone. 1981a. Asbestos-cement materials used in water supply. Water Eng. Manage. 128:48-60.

Millette, J.R., R.L. Boone, M.T. Rosenthal, and L.J. McCabe. 1981b. The need to control asbestos fibers in potable water supply systems. Sci. Tot. Environ. 18:91-102.

Moore, M.R. 1980. Prenatal exposure to lead and mental retardation. In Low Level Lead Exposure: The Clinical Implications of Current Research. L. Needleman, ed. Raven Press, New York.

Moore, M.R., P.A. Meredith, B.C. Campbell, and A. Goldberg. 1977. Contribution of lead in drinking water to blood-lead. Lancet 2:717-718.

Muniz, I.P. and H. Leivestad. 1980. Toxic effects of aluminum on the brown trout, Salmo trutta L. pp. 320-321. In D. Drablos and A. Tollan, eds. Ecological Impact of Acid Precipitation. SNSF Project, Oslo, Norway.

National Academy of Sciences. 1980. Lead in the human environment. National Academy of Sciences, Washington, DC.

Needleman, H.L., C. Gunnoe, A. Leviton, R. Reed, H. Peresie, C. Maher and P. Barrett. 1979. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. New Eng. J. Med. 300:688-695.

Nordberg, G.F., and P. Strangert. 1978. Fundamental aspects of dose-response relationships and their extrapolation for non-carcinogenic effects of metals. Environ. Health Perspect. 22:97-108.

Norseth, T. 1979. Aluminum, pp. 275-281. In Handbook on the Toxicology of Metals. L. Friberg, C.F. Nordberg, and V.B. Vouk, eds. Elsevier, New York.

Norton, S.A., Davis, R.B., and Brokhu, D.F. 1981a. Responses of northern New England lakes to atmospheric inputs of acids and heavy metals: Comp. Rep. A-048ME, U.S. Dept. Int. 90 p.

Norton, S.A., C.T. Hess, and R.B. Davis. 1981b. Rates of accumulation of heavy metals in pre- and post-European sediments in New England lakes. pp. 409-421 In Atmospheric Pollutants in Natural Waters, S.J. Eisenreich, ed. Ann Arbor: Ann Arbor Science Publishers, Ann Arbor, Michigan.

Norton, S.A., T.A. Haines, and J.S. Kahl. 1984. The potential for health effects from acidic precipitation-induced changes in water supplies. 10th Annu. Maine Biol. Med. Sci. Symp., Univ. Maine, Orono.

Nriagu, J.O. 1980. In Cadmium in the Environment. J.O. Nriagu, ed. John Wiley & Sons, New York. pp. 71-114.

Nutrition Foundation's Expert Advisory Committee. 1982. Assessment of the safety of lead and lead salts in food. The Nutrition Foundation, Inc., 489 Fifth Avenue, New York 10017.

Oliver, T. 1911. A lecture of lead poisoning and the race. Brit. Med. J. 1:1096-1098.

Olson, K.R., H.L. Bergman, and P.O. Fromm. 1973. Uptake of methyl mercuric chloride and mercuric chloride by trout: A study of uptake pathways into the whole animal and uptake by erythrocytes in vitro. J. Fish. Res. Board Can. 30:1293-1299.

Ondreicka, R., E. Ginter, and J. Kortus. 1966. Chronic toxicity of aluminum in rats and mice and its effects on phosphorus metabolism. Br. J. Ind. Med. 23:305-312.

O'Shea, T.A., and K.H. Mancy. 1978. The effect of pH and hardness metal ions on the competitive interaction between trace metal ions and inorganic and organic complexing agents found in natural waters. Water Res. 12:703-712.

Ouellet, M., and H.G. Jones. 1983. Paleolimnological evidence for the long-range atmospheric transport of acidic pollution and heavy metals into Quebec, Canada. Can. J. Earth Sci. 20:23-26.

Pacyna, J.M., A. Semb, and J.E. Hanssen. 1984. Emission and long-range transport of trace elements in Europe. Tellus 36B:163-178.

Pagenkopf, G.K. 1983. Gill surface interaction model for trace-metal toxicity to fishes: role of complexation, pH, and water hardness. Environ. Sci. Technol. 17:342-347.

Patterson, C.C. 1980. In Lead in the Human Environment. National Academy of Sciences. Washington, DC. pp. 265-349.

Patterson, C., and D. Settle. 1977. Comparative distributions of alkalies, alkaline earths and lead among major tissues of the tuna *Thunnus alalunga*. Marine Biol. 39:289-295.

Peirson, D.H., P.A. Cawse, L. Salmon, and R.S. Cambray. 1973. Trace elements in the atmospheric environment. Nature 241:252-256.

Perl, D.P., D.C. Gajdusek, R.M. Garruto, R.T. Yanagihara, and C.J. Gibbs, Jr. 1982. Intraneuronal aluminum accumulation in amyotrophic lateral sclerosis and parkinsonism-dementia of Guam. Science 217(4564):1053-1055.

Phillips. G.R.., and D.R. Buhler. 1978. The relative contributions of methylmercury from food or water to rainbow trout (Salmo gairdneri) in a controlled laboratory environment. Trans. Am. Fish. Soc. 107:853-861.

Phillips, G.R., and R.W. Gregory. 1979. Assimilation efficiency of dietary methylmercury by northern pike (Esox lucius). J. Fish. Res. Board Can. 36:1516-1519.

Phillips, G.R., T.E. Lenhart, and R.W. Gregory. 1980. Relation between trophic position and mercury accumulation among fishes from the Tongue River Reservoir, Montana. Environ. Res. 22:73-80.

Piomelli, S. 1980. The effect of low-level lead exposure on heme metabolism, pp. 67-74. In Low Level Lead Exposure: The Clinical Implications of Current Research. H.D. Needlemen, ed. Raven Press, New York.

Piscator, M. 1981. Role of cadmium in carcinogenesis with special reference to cancer of the prostate. Environ. Health Perspec. 40:107-120.

Piscator, M. 1982. Cadmium exposure and effects in the general population and in occupationally exposed workers, pp. 521-536, In Clinical, Biochemical and Nutritional Aspects of Trace Elements. A. Prasad, ed., Alan R. Liss, Inc.

Rahn, K.A. 1981. Relative importances of North America and Eurasia as sources of Arctic aerosols. Atmos. Environ. 15:1447-1445.

Ramamoorthy, S. and D.J. Kusher. 1975. Binding of metal ions in river water (abstract), pp. 19-21. In International Conference on Heavy Metals in the Environment. Toronto, Canada. October 27-31.

Registration Committee, European Dialysis and Transplant Association. 1980. Dialysis dementia in Europe. Lancet 3:190-192.

Reisinger, M., M. Stoeppler, and H.W. Nurnberg. 1981. Evidence for the absence of biological methylation of lead in the environment. Nature (London) 291:228-230.

Reuther, R., R.F. Wright, and U. Forstner. 1981. Distribution and chemical forms of heavy metals in sediment cores from two Norwegian lakes affected by acid precipitation. pp. 318-321 in Proc. 3rd Int. Conf. on Heavy metals in the Environment. Amsterdam.

Rodgers, D.W., and S.U. Qadri. 1982. Growth and mercury accumulation in yearling yellow perch, Perca flavescens, in the Ottawa River, Ontario. Environ. Biol. Fish. 7:377-383.

Rodgers, D.W., and F.W.H. Beamish. 1983. Water quality modifies uptake of waterborne methylmercury by rainbow trout, *Salmo gairdneri*. Can. J. Fish. Aquat. Sci. 40:824-828.

Roels, H.A., J.P. Buchet, A. Bernard, J. Hubermont, R.R. Lawerys, and P. Masson. 1978. Investigations of factors influencing exposure and response to lead, mercury and cadmium in man and animals. Environ. Health Perspect. 25:91-96.

Roels, H., R. Lauwerys, and A.N. Dardenne. 1983. The critical level in renal cortex: A reevaluation. Toxicol. Lett. 15:357-360.

Rosen, J.F., R W. Chesney, A. Hamstra, H.F. DeLuca, and K.R. Mahaffey. 1980. Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. New Eng. J. Med. 302:1128-1131.

Rudd, J.W.M. personal communication. Freshwater Institute, Department of Fisheries and Oceans, Winnipeg, Manitoba, Canada.

Rudd, J.W.M., A. Furutani, and M.A. Turner. 1980. Mercury methylation by fish intestinal contents. Appl. Environ. Microbiol. 40:777-782.

Rudd, J.W.M., M.A. Turner, A. Furutani, A.L. Swick, and B.E. Townsend. 1983. The English-Wabigoon River system: I. A synthesis of recent research with a view towards mercury amelioration. Can. J. Fish. Aquat. Sci. 40:2206-2217.

Ryan, J.A., H.R. Pahren, and J.B. Lucas. 1982. Controlling cadmium in the human food chain: A review and rationale based on health effects. Environ. Res. 28:251-302.

Sangalang, G.B., and H.C. Freeman. 1979. Tissue uptake of cadmium in brook trout during chronic sublethal exposure. Arch. Environ. Contam. Toxicol. 8:77-84.

Scheider, W.A., D.S. Jeffries, and P.J. Dillon. 1979. Effects of acidic precipitation on Precambrian freshwaters in southern Ontario. J. Great Lakes Res. 5:45-51.

Scherer, E., F.A.J. Armstrong, and S.H. Nowak. 1975. Effects of mercury-contaminated diet upon walleyes, *Stizostedion vitreum vitreum* (Mitchill). Canada Department of Environment, Fisheries and Marine Service Research and Development Technical Report No. 597, Freshwater Institute, Winnipeg, Manitoba, Canada.

Schindler, D.W., R.H. Hesslein, R. Wagemann, and W.S. Broecker. 1980. Effects of acidification on mobilization of heavy metals and radionuclides from sediments of a lake. Can. J. Fish. Aquat. Sci. 37:373-377.

Schindler, D.W., and M.A. Turner. 1982. Biological, chemical and physical responses of lakes to artificial acidification. Water Air Soil Pollut. 18:259-271.

Schofield, C.L., and J.R. Trojnar. 1980. Aluminum toxicity to brook trout (*Salvelinus fontinalis*) in acidified waters, pp. 341-362. *In* Polluted Rain. T.Y. Toribara, M.W. Miller, and P.E. Morrow, eds. Plenum Press, New York.

Semple, A.B., W.H. Parry, and D.E. Phillips. 1960. Acute copper poisoning. Lancet 2:715-720.

Shahristani-al, H. and K.M. Shihab. 1974. Variation of biological half-life of methylmercury in man. Arch. Environ. Health 27:342.

Sharrett, A.R., A.P. Carter, R.B. Orheim, and M. Feinleib. 1982. Daily intake of lead, cadmium, copper and zinc from drinking water: The Seattle study of trace metal exposure. Environ. Res. 28:251-302.

Shin, E.B., and P.A. Krenkel. 1976. Mercury uptake by fish and biomethylation mechanisms. J. Water Pollut. Control. Fed. 48:473-501.

Shore, D., and R.Y. Wyatt. 1983. Aluminum and Alzheimer's disease. J. Nerv. Ment. Dis. 171:553-558.

Sloan, R., and C.L. Schofield. 1983. Mercury levels in brook trout (*Salvelinus fontinalis*) from selected acid and limed Adirondack lakes. Northeast. Environ. Sci. 2(3/4):165-170.

Smith, W.H., and T.G. Siccama. 1981. The Hubbard Brook ecosystem study: biogeochemistry of lead in the northern hardwood forest. J. Environ. Qual. 10:323-333.

Spencer, H., C.R. Asmussen, R.B. Holtzman, and L. Kramer. 1979. Metabolic balances of cadmium, copper, manganese and zinc in man. Am. J. Clin. Nutr. 32:1867-1875.

Sposito, G. 1981. Trace metals in contaminated wastes. Environ. Sci. Technol. 15:396-403.

Steffan, R.S., and M.R. Winfrey. 1984. Effect of experimental acidification on mercury methylation and volatilization in a northern Wisconsin lake. Abst. Annu. Meeting Am. Soc. Microbiol. 84:207.

Stock, A., and F. Cucuel. 1934. Propagation of mercury. Die Naturwissenschaften 22:390-393.

Stokes, P.M. unpublished data. Institute for Environmental Studies, University of Toronto, Toronto, Ontario, Canada.

Stokes, P.M., S.I. Dreier, M.O. Farkas, and R.A.N. McLean. 1983. Mercury accumulation by filamentous algae: a promising biological monitoring system for methylmercury in acid-stressed lakes. Environ. Pollut. (B) 5:255-271.

Suns, K., C. Curry, and D. Russell. 1980. The effects of water quality and morphometric parameters on mercury uptake by yearling yellow perch. Ontario Ministry of the Environment, Technical Report LTS 80-1, Rexdale, Ontario, Canada.

Swedish Expert Group. 1971. Methylmercury in fish - a toxicological-epidemiological evaluation of risks. Report from an expert group. Nordisk. Hyg. Tidskript. Suppl. 4.

Taylor, F., J.A. Taylor, G.E. Symons, J.J. Collins, and M. Schock. 1984. Acid precipitation and drinking water quality in the eastern United States: 600/2-84-054. U.S. Environmental Protection Agency, Cincinnati, Ohio. 195 p.

Thompson, J.A.J., and J.A. Crear. 1980. Methylation of lead in marine sediments. Mar. Pollut. Bull. 11:251-253.

Tomlinson, G.H., R.J.P. Brouzes, R.A.N. McLean, and J. Kadlecek. 1980. The link between acid precipitation, poorly buffered waters, mercury and fish. p. 134-137. In D. Drablos and A. Tollan (ed.). Ecological impact of acid precipitation. SNSF Project, Norwegian Interdisciplinary Research Programme. Oslo, Norway.

Tsai, S.C., G.M. Boush, and F. Matsumura. 1975. Importance of water pH in accumulation of inorganic mercury in fish. Bull. Environ. Contam. Toxicol. 13:188-193.

Turner, M.A., and A.L. Swick. 1983. The English-Wabigoon River System: IV. Interaction between mercury and selenium accumulated from waterborne and dietary sources by northern pike (*Esox lucius*). Can. J. Fish. Aquat. Sci. 40:2241-2250.

Tyler, G. 1978. Leaching rates of heavy metals ions in forest soil. Water Air Soil Pollut. 9:137-148.

Tyler, G. 1981. Leaching of metals from the A-horizon of a spruce forest soil. Water Air Soil Pollut. 15:353-369.

U.S./Canada Memorandum of Intent on Transboundary Air Pollution. February 1983. Working Group I. Final Report.

U.S. Department of Commerce. 1978. Report on the chance of U.S. seafood consumers exceeding the current acceptable daily intake for mercury and recommended regulatory controls. National Marine Fisheries Service, Seafood Quality and Inspection Division, U.S. Department of Commerce, Washington, DC.

U.S. Environmental Protection Agency. 1979. Water quality criteria: Asbestos. Federal Register 44(191):56632-56635.

U.S. Environmental Protection Agency. 1979a. The health and environmental impacts of lead and an assessment of a needs for limitations. U.S. Department of Commerce, National Technical Information Service, PH-296-903.

U.S. Environmental Protection Agency. 1979b. National secondary drinking water regulations. Federal Register 44:140.

U.S. Environmental Protection Agency. 1980a. Ambient water quality criteria for mercury. EPA 440/5-80-058. Office of Water Regulation and Standards, Criteria and STandards Division, Washington, DC.

U.S. Environmental Protection Agency. 1980b. Ambient water quality criteria for lead. Office of Water Regulation and Standards. Criteria of Standards Division, Washington, DC.

U.S. Environmental Protection Agency. 1983. National revised primary drinking water regulations: advance notice of proposed rulemaking. Federal Register 48(194):45502-45521.

Wesley, M.L., B.B. Hicks, W.P. Dannevik, S. Frisella, and R.B. Husar. 1977. An eddy-correlation measurement of particulate deposition from the atmosphere. Atmos. Environ. 11:561-564.

Wesley, M.L., and B.B. Hicks. 1978. High frequency temperature and humidity correlation above a warm wet surface. J. Appl. Meterol. 17:123-128.

Westernhagen, H.V., V. Dethlefsen, and H. Rosenthal. 1980. Correlation between cadmium concentration in the water and tissue residue levels in dab, Limanda limanda L., and plaice, Pleuronectes platessa L. J. Marine Biol. Assos. U.K. 60:45-58.

Wheatley, B. 1979. Methyl mercury in Canada. Medical Services Branch, Ministry of National Health and Welfare, Ottawa, Canada.

Wiener, J.G. 1983. Comparative analyses of fish populations in naturally acidic and circumneutral lakes in northern Wisconsin. U.S. Fish and Wildlife Service, Eastern Energy and Land Use Team, FWS/OBS-80/40.16.

Wong, C.S., and P. Berrang. 1976. Contamination of tap water by lead pipe and solder. Bull. Environ. Contam. Toxicol. 15:530.

Wong, P.T.S., Y.K. Chau, and P.L. Laxon. 1975. Methylation of lead in the environment. Nature (London) 253:263-264.

Wood, J.M. 1980. The role of pH and oxidation-reduction potentials in the mobilization of heavy metals. Pages 223-232 In T.Y. Toribara, M.W. Miller, and P.E. Morrow, editors. Polluted rain. Plenum Press, New York, USA.

World Health Organization. 1972. The hazards of persistent substances in drinking water. World Health Organization, Copenhagen.

World Health Organization. 1976. Environmental health criterial: mercury. World Health Organization, Geneva.

World Health Organization. 1977. Environmental Health Criteria 3. Lead. World Health Organization, Geneva.

Wren, C.D., and H.R. MacCrimmon. 1983. Mercury levels in the sunfish, Lepomis gibbosus, relative to pH and other environmental variables of Precambrian Shield lakes. Can. J. Fish. Aquat. Sci. 40:1737-1744.

Wren, C.D., H.R. MacCrimmon, and B.R. Loescher. 1983. Examination of bioaccumulation and biomagnification of metals in a Precambrian Shield lake. Water Air Soil Pollut. 19:277-291.

Wright, D.R., and R.D. Hamilton. 1982. Release of mercury from sediments: effects of mercury concentration low temperature and nutrient addition. Can. J. Fish. Aquat. Sci. 39:1459-1466.

Young, E.S., Jr., and W.E. Sharpe. 1984. Atmospheric deposition and roof-catchment cistern water quality. J. Environ. Qual. 13:38-43.

Yule, W., R.R. Lansdown, I.B. Millar, and M.A. Urbanowicz. 1981. The relationship between blood lead concentration, intelligence and attainment in a school population: a pilot study. Dev. Med. Child Neurol. 23:567-76.

# APPENDICES

## APPENDIX A: AGENDA FOR WORKSHOP

Research Needs Regarding Trace Contaminants and Acid Deposition
as Related to Increased Human Exposure

Top Ridge Conference Center
Paul Smiths, New York

June 19-22, 1984

## AGENDA

### Tuesday, June 19

| | |
|---|---|
| 5:00 pm | Arrival at Top Ridge Conference Center. Transportation will be provided from the Albany, NY airport. |
| 5:30 - 7:30 pm | Reception and Dinner at Top Ridge Lodge. |
| 7:30 - 9:00 pm | Opening remarks and workshop organization. |

### Wednesday, June 20

| | |
|---|---|
| 7:30 am | Breakfast at Top Ridge Lodge. |
| 8:00 am - Noon | Plenary session. Introduction of participants and informal presentations. |
| Noon - 1:00 pm | Lunch at Top Ridge Lodge. |
| 1:00 - 5:30 pm | Review and development of state-of-the-art summaries concerning trace contaminants and acid deposition. Discussions will take place in working groups focusing on atmospheric processes and ecosystem effects leading to increased human exposure. |
| 6:30 - 7:30 pm | Dinner at Top Ridge Lodge. |

Thursday, June 21

| | |
|---|---|
| 7:30 am | Breakfast at Top Ridge Lodge |
| 8:00 am - Noon | Further discussion in working groups of research needs and the development of working group reports. |
| Noon - 1:00 pm | Lunch at Top Ridge Lodge |
| 1:00 - 3:00 pm | Break |
| 3:00 - 6:30 pm | General session, including working group reports, general discussion and development of workshop recommendations. |
| 7:30 - 8:30 pm | Dinner at Top Ridge Lodge. |

Friday, June 22

| | |
|---|---|
| 7:30 am | Breakfast at Top Ridge Lodge. |
| 8:00 am | Adjournment. |
| Noon | Approximate arrival time at Albany, NY airport. |

# APPENDIX B: ACKNOWLEDGEMENTS

The editors thank the workshop participants for their enthusiasm, diligence and thoroughness in the development and review of this report. Continual interest and encouragement have been provided by Joseph M. Eilers, U.S. EPA. His patience and constructive input throughout the review process are greatly appreciated. Finally, special recognition is given to supporting staff, Sharon Hotaling and Denise Polsinelli, who worked long, tedious hours in preparation of this report.